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ARCHIVES OF OPHTHALMOLOGY.

PURULENT CONJUNCTIVITIS IN INFANTS UNDER TWO MONTHS OF AGE.[1]

INCIDENCE, CAUSES, AND EFFECT ON VISION.

BY ANNA W. WILLIAMS, M.D., AND CAROLYN ROSENBERG, M.D., NEW YORK.

THE investigations reported in this paper were undertaken by the Bureau of Laboratories of the N. Y. City Health Department, incited chiefly by the Committee for the Prevention of Blindness, in order to help determine the present incidence, causes, and results of purulent conjunctivitis in infants under two months of age.

We are calling the condition investigated *purulent conjunctivitis in infants* rather than ophthalmia neonatorum, because even yet, notwithstanding the admirable work of Stephenson and others calling attention to the fact that several micro-organisms besides the gonococcus might produce this condition, the majority of physicians consider ophthalmia neonatorum and gonococcus infection of the conjunctiva in the newborn as synonymous.

We thought if we could bring out further statistics to show that this opinion is incorrect, it might help do away with the feeling of reluctance to report such cases to the Health Department.

Incidentally we wished to find out how many cases of this type were not reported to the Health Department.

[1] Presented at the meeting of the Section on Ophthalmology, New York Academy of Medicine, November, 1915.

We desired further to determine the number of cases in which preventive treatment was not used and the effect on the course of the cases.

The investigations are still incomplete, but we thought the first report might be given at this time.

INCIDENCE OF PURULENT CONJUNCTIVITIS IN INFANTS.

The question of the incidence is being studied by securing data in relation to the condition of the eyes of 1000 consecutive births in two definite districts, one in the poorer part of the city, the other in the more well-to-do. The first district chosen is between 14th Street south to the river, and between 3d Avenue and East River. The investigation in this district has just been completed. The children born on the 1st of December, 1914, and successively thereafter, have been visited. Forty-two cases were not seen, because of removal or wrong addresses. Hence, the 1000 cases reported here are among the first 1042 births.

Fifteen babies died, two of whom gave history of purulent conjunctivitis. Six children, still living, had purulent conjunctivitis, two of which were probably due to infection with the gonococcus. All were treated and have eyes now normal. Fourteen children were seen who had conjunctivitis with no history of purulent discharge and had normal eyes at time of visit. Nine hundred and seventy-eight cases, then, in the thousand had normal conjunctivas.

These statistics may be arranged in tabular form as follows:

Condition.	Cases.	Percentage.
History of normal eyes.....	978	97.8
" " purulent conjunctivitis.....	8	.8
" " conjunctivitis without pus.....	14	1.4

In all of the conjunctivitis cases the prophylactic treatment had been given. One only of the eight cases was reported. These were midwife cases. In the record card we noted date of birth, prophylactic treatment, name and address of obstetrician, date of onset, symptoms, treatment, when and by whom treated, sources of infection, present condition of eyes.

CAUSES OF PURULENT CONJUNCTIVITIS IN INFANTS.

Data and cultures were obtained from a series of cases reported to the Health Department chiefly by the midwife service.

From November 17, 1914, to July 31, 1915, ninety-one cases in all were reported to the Health Department; forty-three were found to be either treated by physicians (seventeen cases), or were incorrectly reported, the eyes being normal. There remain forty-eight cases from whom data in relation to signs, onset, possible prophylactic treatment, possible source of infection, were secured and from forty-seven of which cultures were taken.

For the study of the microörganisms, the following is our method of procedure:

1. (*a*) Note is made of the appearance of the eyes; (*b*) whether a discharge be present or not; if present, the character and amount of it; (*c*) the condition of the eyelids, whether œdematous or inflamed or encrusted; (*d*) the condition of the conjunctiva lining the lids, and of that covering the eyeball; (*e*) the condition of the cornea.

2. The conjunctivas lining the lids of the eyes are gently curetted. Smears are made from one curettement, and as soon as dried, by the air, are fixed in Methyl Alcohol. The material from the next curettement is placed in tubes of blood-broth. The next is placed in a small amount of ascitic fluid. Each eye is then washed with boric acid.

3. The mother is advised as to what should be done and referred to the nearest eye dispensary.

4. The smears are stained by the Giemsa method and by Gram.

5. Blood-drop agar plates and plates of acid agar (Sabouraud's medium) are inoculated by a loopful of material from the blood-broth.

6. From the ascitic fluid small amounts are placed in tubes of kidney ascitic fluid. Different dilutions are made. Each is covered by a layer of sterilized petroleum and incubated anaerobically. These are examined on about the fourth, the tenth, and the fifteenth days. Smears are made and stained by the Giemsa method. Stab inoculations are made in deep narrow

tubes of semi-solid ascitic agar. Aërobic and anaërobic plates are made.

From the original blood broth and from the blood-drop agar, transplants on other media, and smears are made until identification of the different organisms is established.

The acid agar medium is used for determining the presence of molds and yeasts.

By our method of using enriching media for the curetted material we are reasonably sure that if living gonococci are present, we can detect them.

Organisms Found.—The following table shows the incidence of the bacteria found in the forty-seven cases of purulent conjunctivitis from which cultures were made.

Microörganism.	*In No. of Cases.*	*Percentage.*
Staphylococcus	47	100.
Xerosis	42	89.3
Pneumoc.-streptococcus group	22	46.3
Gonococcus	12	25.5
[1] Influenza bac. group	12	25.5
Morax-Axenfeld	3	6.3
Sarcinæ	3	6.3
Bac. subtilis	1	2.1

In two cases only—4.2%—were inclusion ("trachoma inclusion") bodies found. In one case—2.1%—indefinite bodies, which somewhat resembled inclusion bodies, were seen.

Staphylococcus and bac. xerosis were commonly found, but usually in small numbers. Only in one fourth of the cases was the gonococcus definitely found to act as an ætiological factor. Twenty cases where no gonococci were found have been considered as of possible gonococcus origin because of early date of onset, and time elapsing between onset and date of first visit. These twenty cases all made a good recovery.

The pneumoc.-streptococcus group were demonstrated about twice as often as were the gonococcus and the B. influenzæ group. The two last mentioned groups of organisms were found in an equal number of times in this series of cases.

In relation to the streptococcus-pneumococcus group, in

[1] Koch-Weeks bacillus of ophthalmology.—Ed.

six consecutive cases in which they appeared, they were of the hæmolytic type; while the following thirteen cases, which were of the viridens type, suggested the possibility of contamination from two distinct types of infection, which may have been prevalent during these two respective periods.

In the anaërobic cultures no microörganism was found which had not appeared in the aërobic cultures.

1. *Date of Onset.*—The *ages* of the infants at the time of the first visit, which was usually within twenty-four hours of the reporting of the case, were from three to forty-seven days.

11	were seen during	1st	week
23	" " "	2d	"
8	" " "	3d	"
4	" " "	4th	"
1	was " "	5th	"
1	" " "	7th	"

The *onset* of the disease took place as follows:

4 or 8.3% at birth
28 " 58.3% during first 3 days
35 " 72.9% " " week
10 " 20.8% " second week
6.3% unknown (as they were "foundlings" brought to the Manhattan Eye and Ear Hospital).

2. The *onset* of the cases where the following bacteria were found was as follows:

Gonococcus, from birth to the 14th day — 1 at birth; 4 during 1st week; 2 " 2d " = 6 cases

Streptococcus-pneumococcus group, from birth to 10th day — 1 at birth; 11 during 1st week; 1 " 2d " = 12 "

Influenza group, from 2d to 11th day — 3 during 1st week; 2 " 2d " = 5 "

Gonococcus and *streptococcus-pneumococcus groups* — 1 on 2d day = 1 " 13th " = 1 during 1st week; 1 " 2d " = 2 "

Influenza and *streptococcus-pneumococcus groups*, from birth to 9th day — 2 at birth; 5 during 1st week; 1 " 2d " = 6 "

The *gonococcus*, the *influenza*, and the *streptoc.-pneumococcus groups*, only one, which occurred on the seventh day or during the first week. The remaining ten cases of *staphylococcus* and *xerosis bacillus* infection from third to thirteenth day, eight during first week and two during second week.

3. *Use of Prophylactic Treatment.*—In thirty-seven cases, or 77%, it was determined through mothers' statements that a prophylactic was used.

Mothers' statements, however, cannot be accepted as reliable. In all probability there was a higher percentage than 77% which had received prophylaxis.

After Treatment.—In thirteen cases there had been no treatment up to time of seeing infant.

In thirty-four cases boric acid had been used by mother, and among these, where infant had been in hospital or to dispensary, argyrol also had been used.

In three cases, mother used breast milk, thinking it was beneficial.

With the exception of one midwife, who reported Cases 37 and 38, there was not more than one case from the same midwife.

Possible Source of Infection.—It was impossible to elicit sufficient history of the families and of those who had come in contact with the infants, to get sufficient data to point toward a possible source of infection in each of the cases. The following eleven cases were the only ones in which probable associative infection could be found.

In Case 13—where influenza bacilli were found—onset eighth day—two other children had coryza and bronchitis.

In Case 19—where influenza bacilli were found—several other children of family had "colds."

In Case 26—where a streptococcus culture was obtained—nurse, with whom baby slept, had "cold."

In Case 27—where influenza bacilli were found—mother had "cold."

In Case 28—where influenza bacilli were found—child died from "cough." He had had a "cold" soon after birth.

In Case 29—where streptococci were found—another child had nasal discharge and "cold."

In Case 31—where streptococci were found—mother had "cold."

In Case 32—where streptococci and influenza bacilli were found—mother and another child had "colds."

In Case 35—where streptococci and influenza bacilli were found—another child in family had "cold."

In Case 45—where gonococcus was found—(onset fourth day) mother had a profuse vaginal discharge. Another child, now two and a half years, with eyes now normal, had had purulent conjunctivitis.

Defective Vision Resulting from Ophthalmia Neonatorum.—In determining the relationship of defective vision to purulent conjunctivitis, we have noted (1) the organisms found in these cases; (2) the date of onset; (3) the relation of onset to the use of prophylactic and curative treatment, and (4) the questions of source and complications of infection.

So far we have found only three cases where vision is affected, less so in one than the other two. We are keeping these cases under observation. In all of them gonococci, staphylococci, and xerosis bacilli were found. In all, prophylactic treatment had been used. The onset of one was on the thirteenth day; of another, on the fourth day; and the third was on the first day. In the first case, the source of infection was not determined; in the second, the mother had clinical gonorrhea; and in the third, source undetermined.

SUMMARY AND CONCLUSIONS.

1. In a thousand consecutive births only 0.8% had purulent conjunctivitis.

2. In ten cases out of forty-seven cultured cases or in 21.2% only staphylococcus or staphylococcus and the xerosis bacillus were found, but not in large enough numbers to be considered a cause.

3. In twelve of these cultured cases, or in 25.5%, the gonococcus was found. In nine of these twelve cases, or in 19.1% only, of all the cases, was the gonococcus found without the presence of either the streptococcus-pneumococcus group or the influenza group of organisms. In three of these twelve cases, or in 6.3% of all the cases, the gonococcus was associated with either or both the streptococcus-pneumococcus and the influenza groups.

4. In twenty-two out of forty-seven cultured cases, or in 46.3% of all the cases, was the streptococcus-pneumococcus group of organisms found independent of the presence of the gonococcus and the influenza bacillus. It was thus found to be apparently about twice as often a cause of purulent conjunctivitis as the gonococcus.

5. In twelve cases, or in 25.5% (the same number of times as gonorrheal infection), was the influenza bacillus group found as the apparent exciting cause.

6. A history of 77% of cases positively receiving prophylactic treatment, and of a probably greater percentage having received it, suggests the advisability of treatment on the second day with $AgNO_3$ solution.

7. In only three cases was the cornea involved. The fact that these three cases were gonorrheal infections confirms the opinion that cases of purulent conjunctivitis which are infected with this organism are liable to be more severe in infants than those due to other infections.

8. Purulent conjunctivitis in infants is due, in one third of the cases at least, to organisms other than the gonococcus.

9. The fact that purulent conjunctivitis or ophthalmia neonatorum does not always mean an infection with the gonococcus should make physicians more ready to report such cases to the Health Department.

DIGITAL COMPRESSION OF THE LACRIMAL SAC IN DACRYOCYSTITIS, ESPECIALLY OF THE NEWBORN.

By Dr. JUAN SANTOS-FERNANDEZ, Havana, Cuba.

THE diseases of the lacrimal ducts, studied for many years and recently perfected, offer some interesting, if not new, aspects to the clinician. Because of the rarity with which an especial pathological manifestation presents itself, it is necessary to fix our attention upon something that may be beneficial, yet on account of its simpleness may pass unobserved, such as the massage of the lacrimal sac in congenital dacryocystitis given by digital compression and continuously.

When we saw our first case of congenital dacryocystitis, we were in doubt concerning the procedure to be adopted by us. The difficulty of surgical intervention in a newborn induced us to employ the manual treatment in this case, and as a good result was obtained on it, we kept on using digital compression of the sac, even in the adult, with similar results where the ætiology had been different.

Compression of the lacrimal sac needs ability, as is the case with all manual procedures, and requires practice, for otherwise one imagines that the sac has been compressed when it is not so; this may happen especially if the vicinity of the sac is inflamed or œdematous. When the affection is uniocular, with the unaffected side as a guide one should try to find the crest of the os unguis, or lacrimal bone, and then to direct the pressure behind that crest, because the lacrimal sac should be looked for in the fossæ at that place.

When the compression is made in order to make a diagnosis, and is then carried out by the physician, he should bear in

mind these details, and also he should try to ascertain if the canal is closed or open, because in the first case the contents of the sac pass to the nose or fauces. If the compression is ordered as a treatment and it is performed by the patient or some relative, we can not be sure of its being done in the proper way.

In order to know the permeability of the nasal ducts, it has been sufficient for us to drop a small quantity of a solution of fluorescein, because if there is a stenosis the swab of cotton applied to the nose, on blowing it, will not be stained with that solution. The aforesaid exploration, that looks futile, is omitted more than once, as has been proved in our practice. Some patients in which we had not made the catheterism of the naso-lacrimal ducts because there had been the passage of the water through it to the nose and throat, have gone away and had been treated in other places with Bowman's probes, and when those patients have come back to us, still with the same permeability, we have asked ourselves the reason for using catheterism in them.

Catheterism is not needed then for, as Berard of Angoulême has said,[1] what is needed is to cure the dacryocystitis, a thing that we have obtained frequently by using the compression of the sac and the antiseptic injections through the lacrimal punctæ, which had been previously dilated.

There is sometimes a blind desire for practicing the catheterism of the nasal ducts, which should be performed only by those that have experience, otherwise the obstacle will not be overcome. According to a noted practitioner, Dr. E. v. Hippel of Halle, who says: "The probing of the nasal duct is not easy nor innocuous. He who is not accustomed to do it will construct new ways that will produce new scars."

More than once we have spoken about the abuse of the catheterism of the lacrimal ducts,[2] declaring ourselves against those who imagine that all the treatment consists in is the probing of the lacrimal ducts even in a congenital dacryocystitis. In newborn, this is due, more often (*cf.* Hippel), to

[1] *Société française d'ophtalmologie.* Séance du Mai 4, 1905.

[2] "On the Abuse of Catheterism of the Nasal Duct." *Anales de Oftalmología de México,* vol. xiii., p. 325. Also in *The Crónica Médico-Quirúrgica de la Habana,* vol. xxxvi., p. 413.

the occlusion of the lower exit of the naso-lacrimal canal, and a vigorous pressure upon the sac will break the membranous occlusion.

Coppez, also, has cured congenital dacryocystitis by the simple digital compression of the sac, followed by antiseptic injection, as affirmed by Parent, and Chevalleraux too has employed digital pressure and believes that congenital dacryocystitis is due to an imperforation of the nasal canal in its lower part.[1] Feger believes[2] that dacryocystitis of the newborn has not received the care it should have in Germany, and that it is due to an anomaly of the development and is not of gonococcal origin. He recommends catheterism if massage has not been enough to re-establish the permeability of the duct.

Pechin believes that congenital dacryocystitis is due to an obstruction of the nasal duct, caused by epithelial casts, which in turn are due to the staphylococcus, habitual microbial agent. The cure he thinks can be spontaneous, or by massage and injections or catheterism.[3]

Terson says that wet nurses often cure dacryocystitis in the newborn[4] by sucking, or nasal aspiration. He has cured it frequently by digital compression and by instilling nitrate-of-silver solution between the eyelids.

Fage,[5] who has given much time to the dacryocystitis of the newborn, observes that it is often due to occlusion, total or partial, of the lower extremity of the duct, but that this is not the only cause. There exist the anomalies of development of the punctæ, the excretory ducts, the exostosis, and the folds of the mucous membrane of the duct, all of which may produce the obstruction; there being, besides, another factor that is often not taken in mind and which is the passage of infection from the conjunctiva to the duct and nose. In each case he claims it may be cured by digital pressure.

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[1] *Société d'ophtalmologie de Paris*, January, 1898.

[2] *Gygyaszat*, 50 de 1905. *Ann. d'Oculistique*, vol. cxxvi., p. 338. *Archiv für Augenheilkunde*, vol. lviii., fasc. i. *Annales d'Oculistique*, vol. cxxxix., p. 130.

[3] *Annales d'Oculist.*, vol. cxxxi., p. 446. *Arch. d'opht.*, p. 490, 1905, Paris.

[4] *Chirurgie oculaire*, 1901, p. 157.

[5] *Arch. d'opht.*, vol. xxxi., October, 1911. *Société franç. d'opht.*, Mai, 1911.

Cassimatis,[1] attributes congenital dacryocystitis to the anatomical disposition of the naso-lacrimal duct (undue dilatation of the lacrimal ducts), and believes the disease to be of staphylococcic origin. He employs physiological serum in injections.

Antonelli[2] has observed five cases of dacryocystitis (congenital) due to delayed embryonic canalization, and recommends pressure or massage by the mother, after probing by the doctor.

Kin[3] adds to massage the yellow oxide ointment.

Hirsch[4] used in five cases the digital compression, refusing the catheterism. He thinks the disease is due to the vaginal secretion, as in ophthalmia neonatorum, but does not deny the possibility of the incomplete impermeability of the nasal opening of the lacrimal duct.

Zelenkousky[5] proved in a girl three days old the presence of staphylococcus pyogenes aureus, very virulent, in which the volume of the sac was such that it was difficult to make the pus leave the sac through the punctæ, and an incision was necessary.

Elliasberg[6] observed at Salonica, Greece, two cases of congenital dacryocystitis in newborn, that in ten days were cured by digital compression, practiced daily. Koenig cured a case with only one injection, but for another patient he had to make the catheterism. On that account Morax makes a plea to establish a distinction, because there are no proofs to sustain that congenital dacryocystitis is always due to an imperforation of the nasal duct, or to disturbances during the development of this duct, and he believes that it should be more rational to admit the existence of an inflammation of the mucous membrane of the lacrimal duct appearing a few days after birth.

We have given some time to the affections of the lac-

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[1] *La clinique ophtalmologique*, p. 417, 1911.

[2] *Archiv de Oftal. Hispano-Amer.*, Enero, 1906.

[3] "Zur Kasuistik der Dakryocystitis," *Woch. Ther. u. Hyg. d. Auges*, 11 Aôut. 1904.

[4] "Zur Dakryocystitis Congenita," *Arch. Augenh.*, vol. xlv., p. 291.

[5] *Westnik Ophthalmology*, No. 1, 1902.

[6] *Westnik Ophthalmology*, 1900, No. 4–5.

rimal apparatus, and we have stated more than once that on our return to Cuba from Europe we were surprised to find the small number of diseases of the lacrimal apparatus as compared with those we had seen in the eye clinics of Paris and Madrid. We attributed that fact to the large percentage of negro population. To prove this we later on prepared skulls,[1] together with Professor J. A. Presno, Anatomy Professor of Havana University.

The congenital membrane admitted as capable of closing the lower outlet of the naso-lacrimal duct in the newborn, and which causes dacryocystitis, is probably due, very often, to hereditary syphilis.

Having cured a case without antisyphilitic treatment is not sufficient to deny the specific ætiology, because in the clinic we many times see an interstitial keratitis cured without any antisyphilitic treatment,[2] and on its recurrence we have confirmed its specific origin and have established the adequate treatment. Whoever has had a large practice must have had some case of syphilitic iritis that has passed its first attack without specific treatment, or with unwise treatment, and in which there have been no resulting synechiæ, and it is also certain that any attack of iritis, whether it is specific or not, may leave annoying synechiæ, even after well directed treatment.

On returning to our first case of congenital dacryocystitis, it was on a child 55 days old, on which fifteen days after birth

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[1] "Remarks about Diseases of the Eyes in Cuba, in 1875." *Havana Academy of Sciences*, May 14, 1876. *Cron. Med.-Quir.*, vol. ii., p. 551.

"Some Considerations about the Different Races that Inhabit the Island of Cuba." *Havana Anthropological Society*, 1878. *Crónica Oftalmológica de Càdiz*, p. 212, 1879.

"Statistics of the Diseases of the Eyes in Negroes and Mulattoes." *Thirteenth International Congress of Medicine, Ophthal. Sect.*, Paris, August, 1900. *Crónica Médico-Quirúrgica de la Habana*, vol. xxvii., p. 385.

"On the Anatomical Dispositions of the Nasal Duct in the Negro, with Presentation of Skulls." *Fourteenth International Congress of Medicine*, Madrid, 1903. *Archivos de Oftalmologia Hispano-Americanos*, vol. iii., p. 94, 1903.

[2] "Interstitial Keratitis in my Ophthalmic Practice." *Ophthalmology*, vol. x., No. 2, 1914. *Cron.-Med.-Quir.*, vol. xl., p. 144; *Arch. de Oft.*, vol. xiv., p. 1.

there was noticed the bulging of the sac and a secretion that denoted the existence of dacryocystitis. There was also a deformity on the apex of the head, due to an encephaloid tumor which was herniated, and which disappeared by antiseptic washing of the region and constant compression with the finger of the bulging in the lacrimal sac.

The first days, on account of the pressure not being done in the way it was ordered, there was little gain, but when we ordered the parents to insist and to do it properly, so as to avoid an operation later on, they made the methodical compression of the tumor, and when we saw the child some months after, the dacryocystitis had disappeared.

It seems unnecessary to say that when a case has been diagnosed the first thing to be done is to ascertain if the nasal duct is more or less free, because in this case, the thing to do is to treat the dacryocystitis, on curing which the tears would follow their natural course. Without leaving the irrigation undone, the compression should be begun from the first, and any inflammation may be lessened by ice-bags or cold compresses. Under these conditions catheterism should never be done, because it is not only not needed but harmful. We are treating at the present time a lady who when treated by us two years ago for a dacryocystitis was able to return to her city after yielding to the irrigations and the digital compression. As she did not keep on with the irrigations of the sac as ordered she had a new attack a year later, at which time we practiced the dilatation of the lower punctæ, beside the irrigations and pressure on the sac. Again neglecting to follow directions, she suffered a third attack a year after.

We have observed exceptional cases in which in spite of there being dilatation of the lacrimal sac, it has been reduced with digital compression and for ten years there has been only a slight secretion, with no inflammation whatever. One such patient is an old lady that now also has cataracts, and although we have treated similar cases by making use of irrigations of ice-cold water,[1] and with success, we will not repeat the procedure in this case, but shall excise the sac before the extraction of the cataract.

[1] "Antiseptics before and after Cataract Operation (Two Cases of Dacryocystitis)." *Crónica Oftalmológica de Cádiz*, vol. x., p. 13, 1888.

In congenital dacryocystitis, or of the newborn, before ascertaining the cause that has produced it, digital compression should be tried, without forgetting the irrigations, according to circumstances. When both things fail, there is time to employ catheterism and to begin, if necessary, internal or external treatment, if the ætiology should give any light.

ANOMALIES OF THE ACCOMMODATION, CLINICALLY CONSIDERED.[1]

By ALEXANDER DUANE, M.D., New York.

CONSIDERING the amount of attention devoted to refractive errors and their correction, the study of accommodative anomalies has received comparatively little attention from ophthalmologists. Yet those who deal day after day in their consulting rooms with the problems of refraction work recognize, I am sure, that these anomalies often occasion considerable trouble, so that if we fail to diagnosticate and treat them we are not doing our whole duty to our patients.

This neglect of an important subject has been due, we believe, not so much to a failure to recognize its importance, as to the fact that hitherto the precise data on which a satisfactory study should be based have been lacking. For it is evident that before we can say what the symptoms of abnormal accommodation are we must have a clear notion of what we mean by normal accommodation. In other words, we must by defining the limits of normal accommodative action determine when any given accommodation can be called subnormal or supernormal. Up to a few years ago extensive studies on this point were lacking.

This lack the author tried to supply through a series of researches which he has pursued for nine years and reports of which have been made to this society in 1908 (2) and to the Ophthalmological Section of the American Medical Association in 1910 (3) and 1912 (4). Contrary to the impression gathered by some, their main end was different from that sought by

[1] Read in part before the American Ophthalmological Society, July, 1915.

Donders and other predecessors in this field. These observers aimed to obtain a curve representing the mean accommodation at each age. Such a curve, however, is more or less arbitrary, and from the same data one might derive several mean curves (4, 5.) The author's curves and tables showed the mean accommodation and in addition, what is much more important, the maximum and minimum normal accommodation at each age from 10 to 60. As was pointed out in these papers, from these values, which were based on some 12,000[1] measurements made on 1500 cases, one could determine, as one could not from the observations of preceding investigators, whether the accommodation in any given case was normal or not, and, if abnormal, how far it varied from the lower or upper normal limits.

The values obtained are shown in the accompanying table.

TABLE OF NORMAL VALUES OF THE ACCOMMODATIVE POWER AT ALL AGES FROM 8 TO 68

Accommodation is given in D and tenths, near point being measured from the anterior focus of the eye, *i.e.*, from a point 13*mm* in front of the cornea.

Age.	Lower Limit in Normal Cases.	Mean Value.	Usual Upper Limit.	Extreme Upper Limit.
8	11.7	13.8	15.4	16.4
9	11.6	13.6	15.2	16.2
10	11.4	13.4	15.0	16.0
11	11.2	13.3	14.9	15.8
12	11.1	13.1	14.7	15.6
13	10.9	12.9	14.5	15.4
14	10.8	12.7	14.3	15.3
15	10.7	12.6	14.1	15.2
16	10.5	12.4	13.9	14.9
17	10.3	12.2	13.7	14.6
18	10.1	11.9	13.5	14.4
19	9.9	11.7	13.2	14.2
20	9.7	11.5	13.0	14.0
21	9.4	11.2	12 8	13.7
22	9.2	10.9	12.6	13.5
23	8.9	10.6	12.3	13.2

[1] Confirmed by some 3000 taken since.

Age.	Lower Limit in Normal Cases.	Mean Value.	Usual Upper Limit.	Extreme Upper Limit.
24	8.7	10.4	12.1	13.0
25	8.4	10.2	11.8	12.7
26	8.2	9.9	11.6	12.4
27	7.9	9.6	11.3	12.1
28	7.6	9.4	11.1	11.8
29	7.3	9.2	10.7	11.5
30	7.1	8.9	10.4	11.2
31	6.7	8.6	10.2	10.8
32	6.4	8.3	9.9	10.5
33	6.1	8.0	9.6	10.2
34	5.9	7.7	9.2	9.9
35	5.6	7.3	8.9	9.6
36	5.3	7.1	8.6	9.4
37	4.9	6.8	8.2	8.9
38	4.6	6.5	7.9	8.6
39	4.3	6.2	7.6	8.2
40	4.0	5.9	7.2	7.8
41	3.6	5.4	6.8	7.5
42	3.2	5.0	6.4	7.0
43	2.8	4.6	5.9	6.5
44	2.5	4.2	5.5	6.1
45	2.2	3.7	5.2	5.6
46	1.9	3.3	4.8	5.1
47	1.7	2.8	4.3	4.5
48	1.5	2.5	3.9	4.0
49	1.3	2.2	3.4	3.4
50	1.2	2.0	3.0	3.0
51	1.1	1.8	2.6	2.6
52	1.0	1.6	2.3	2.3
53	0.9	1.5	2.1	2.1
54	0.9	1.4	2.0	2.0
55	0.8	1.3	1.9	1.9
56	0.8	1.2	1.8	1.8
57	0.8	1.2	1.7	1.7
58	0.7	1.2	1.7	1.7
59	0.7	1.1	1.6	1.6
60 to 68	0.7	1.1	1.5	1.5

METHOD OF TESTING ACCOMMODATION.

The way in which this table is used is as follows:

The patient whose accommodation we are testing is placed with his back to the light (good, diffuse daylight should be used when possible) and is directed to look sharply at the test-object (a fine black line bisecting a white parallelogram which is mounted on a disk of black velvet). The eye not under examination being covered, the test-object is brought toward the patient along a Prince's rule until the fine line blurs, and then is carried back and forth several times until we ascertain the nearest point at which the line can be seen with perfect distinctness. The position of this nearest point on the rule indicates the accommodation in dioptries.

In order to make the tests comparable with the table, the measurement must be made from the anterior focus of the eye, *i.e.* a point 13*mm* in front of the cornea, or, practically, from the plane of the trial frame or the patient's glasses. Furthermore, the patient must either be provided with his full refractive correction, or if not, a corresponding allowance must be made in the reading found.

Repeated tests should be taken, and care must be exercised that the results are not vitiated by the patient's inattention or lack of comprehension of what is expected of him. Care must be taken, too, that we do not consider as due to failure of accommodation blurred vision caused by insufficient illumination, uncorrected errors of refraction, opacities in the media, or retinal disease.[1] Quite often a very faint and hardly distinguishable central scotoma produces a condition closely simulating an insufficiency of accommodation.

If now, in any given case, repeated tests, properly made, show that the accommodation lies outside of the normal limits given for a person of the patient's age (see table), we may characterize the finding as abnormal.

PRELIMINARY REPORT ON ABNORMAL ACCOMMODATION.

In the papers cited and in one read before the New York State Medical Association in 1913 (6) the general character of the variations from the normal was indicated. The present

[1] For precautions to be observed, see the author's papers (3, 5).

paper aims to give a more precise description of these anomalies, derived from a study of some 170 cases. These do not include all the cases seen, nor is the study offered as being in any sense complete. It is in fact preliminary only.

VARIETIES OF ABNORMAL ACCOMMODATION.

The abnormalities of accommodation that have been observed are as follows:

1. The accommodation may be persistently below the lower normal limit (*Insufficiency of Accommodation*).
2. Akin to this condition and, indeed, often constituting the first stage of it is that in which the accommodation is normal in amount, but is sustained only with effort and soon gives out (*Difficult or Ill-Sustained Accommodation*).
3. There is difficulty or sluggishness in changing from one accommodative state to another (*Inertia of Accommodation*).
4. The accommodation in the two eyes is not the same (*Inequality of Accommodation*).
5. The accommodation lies persistently above the usual normal limit (*Excessive Accommodation*).

INSUFFICIENCY OF ACCOMMODATION.

Definition: By *Insufficiency of Accommodation* we denote the condition in which repeated tests made under proper conditions show that the patient's accommodative power is distinctly and persistently below the normal lower limit for his age (see table). Obviously also a patient must be held to have at least a *relative* insufficiency of accommodation if his accommodative power, although above the minimum normal limit for his age, is persistently below his own lower normal limit as shown by later tests.

Insufficiency of accommodation may be *intermittent* or *constant*, and *transient* or *persistent*. It may also present all grades in intensity. Insufficiency of very high degree (*paresis* and *paralysis* of accommodation) usually differs in origin and character from the moderate insufficiencies and hence will receive separate consideration. On the other hand, difficult and ill-sustained accommodation differs in no essential way

from insufficient accommodation and will be considered with the latter.

Frequency: Insufficiency of accommodation is a fairly frequent affection. Precise statistics on this point are not forthcoming as yet, but the opinion may be hazarded that the condition occurs in at least five per cent. of our patients.

The following table shows the results as regards sex and age incidence.

Age.	Sex. Including all occurring at a given age.		Sex. Excluding those counted twice (*i.e.*, carried on from a previous age period).	
	MALES.	FEMALES.	MALES.	FEMALES.
10–14	3	5	3	5
15–19	17	5	17	5
20–24	10	12	9	11
25–29	9	6	8	6
30–34	14	6	12	6
35–39	6	14	3	14
40–44	6	15	6	13
45–49		6		5
			58	65

The number of cases (123) is too small to warrant sure conclusions, but the following are offered tentatively.

Accommodative insufficiency is not very common before 15 nor after 45. Between 15 and 45 it is about as frequent at one age as another.

From puberty till 20 it occurs three times as often in males as in females.

From 20 to 30 it is about equally frequent in the two sexes.

Between 30 and 35 it is about twice as frequent in males.

After 35 it occurs about three times as often in females as in males.

Types: Insufficiency of accommodation may theoretically be due either to undue rigidity (accelerated sclerosis) of the lens or to weakness of the ciliary muscle.

We believe from our observations that both types actually

exist and can usually be distinguished. In the former type, which may properly be called premature presbyopia and which represents simply a physiological process carried to an undue degree, the accommodation drops in much the normal fashion from year to year, but in any one year is always below the normal standard. The curve of descent, in other words, parallels the normal curve but at a lower level.

In the type due to anomalies of ciliary action, the insufficiency, being due to pathological conditions which are themselves variable, varies from time to time often in a quite haphazard manner, so that the curve of descent exhibits irregular sinuosities and salients. After sinking below normal it may become normal again, which would not be the case in a simple premature presbyopia.

In all probability also mixed cases exist, in which an insufficiency due to impaired ciliary action is added to an insufficiency due to premature sclerosis. These will present the characters of both types.

Associated Muscular Conditions: It would naturally be supposed that an insufficiency of accommodation in which the patient is striving hard to see at near points would be associated with an undue effort of convergence, *i.e.*, with convergence excess. But this association occurred in only a small minority (about 12 per cent.) of our cases.[1] Much more frequent was the association with an often marked but sometimes transient convergence insufficiency (about 44 per cent. of the cases). In some 10 per cent. other motor anomalies were present and in about 34 per cent. the exterior muscles were normal.

Etiology: The causes underlying accelerated sclerosis of the lens and producing simple premature presbyopia are, so far as the author can tell, unknown.

The causes giving rise to ciliary weakness and causing the second (pathological) type of accommodative insufficiency have in our cases been as follows:

1. Toxic conditions due to infectious disorders (intestinal

[1] In these cases of accommodative insufficiency with convergence excess there may be a spasm of accommodation. In other words, the ciliary muscle, weak as it is, may still act in a spasmodic sort of way, just as it does sometimes when partially under the influence of homatropine.

toxæmia, tuberculosis, influenza, whooping cough, measles, tonsillar and probably dental infection).

2. Nasal obstruction. This appears to be a frequent cause especially in the cases associated with convergence insufficiency.

3. Hypopituitarism and other disorders of the internal secretions.

4. Neurasthenia and anæmia, brought on by overwork, impaired nutrition, and similar causes. These cases, too, are prominently associated with convergence insufficiency and sometimes with contracted fields.

5. Vascular hypertension. It is striking to find how often in our notes, even in the case of young people, occurs the statement "vessels engorged and tortuous, arteries sharp, disks much injected." With this condition of things or without is found so often as to indicate its etiological importance a persistent diffuse or reticulate dense injection of the conjunctiva with sometimes follicular or other forms of swelling. These cases are specially apt to be associated with considerable burning and smarting from use of the eyes and with intolerance of light.[1] It has seemed to us not improbable that some of these cases with marked irritative symptoms and photophobia might actually be due to the effect of light on the eyes. One case, at least, seemed to be directly brought on by exposure to electric light.

6. Eyestrain without the other contributing factors and apart from the injurious effect of light is a probable but not apparently a very frequent cause.

Symptoms: The onset may be sudden; even so, apparently, in simple premature presbyopia. In more cases probably the onset is gradual.

The main symptoms are asthenopia—either simple tiring of the eyes or tiring associated with eyeache (sometimes of great severity), with marked irritation and burning of the eyes, or with headache; blurred vision, especially for near work; vertigo; aprosexia; photophobia (quite a marked symptom in a number of cases). These symptoms are often due in part to the causal or associated conditions. Thus the blurred

[1] The not infrequent association of vascular hypertension with asthenopia and conjunctival irritation has been signalized by de Schweinitz.

vision at near points and the vertigo are often due to an associated convergence insufficiency or other muscular trouble; the irritative symptoms to the conjunctival congestion so often present; and the photophobia to this cause or to the retinal congestion.

An occasional finding, especially in the cases with marked convergence insufficiency, is a concentric contraction of the visual field.

The intensity of the symptoms varies greatly. In many cases they are such as to render eye work almost impossible. On the other hand, especially in simple premature presbyopia, the only complaint may be of impaired vision for near, which is remedied at once by a suitable addition to the glasses for reading.

Course and Duration: The course and duration naturally depend on the cause. Recurrences may take place; sometimes at long intervals. In simple premature presbyopia the course goes on much as in an ordinary presbyopia, but the necessity for the use of an additional glass for reading begins much earlier—at forty or before.

Treatment: The treatment is directed at the cause, whenever we can discover the latter. The removal of toxic conditions by remedying an intestinal indigestion or a dental or tonsillar infection; the relief of anæmia and malnutrition by the use of tonics and proper diet; the regulation of vascular anomalies; the removal of nasal obstructions; treatment addressed to abnormal glandular condition—all these are to be employed when required.

Eyestrain naturally should be relieved by proper glasses.

An associated conjunctival injection should be treated with zinc or other astringents.

For the insufficiency itself the following means should be tried:

1. For the blurred vision at near, the distance glass should be supplemented by a proper addition for reading (presbyopic correction for an unnatural presbyopia).

2. Even when the vision is not much blurred, this presbyopic addition may be necessary, especially if there is an associated convergence excess.

3. The accommodation may be stimulated directly by

means of reading exercises or—as I have found useful—by systematic exercise with the fine test-object (line) used in measuring the accommodation. The exercise consists in approximating this to the eye until it blurs and repeating this maneuver several times, trying each time to bring the line closer, and force the eye to accommodate on it. Such exercise can be done two or three times daily.

4. Especially in cases associated with convergence insufficiency it is useful to stimulate both accommodation and convergence by systematic exercise with prisms, bases out, when the eyes are directed at a near object. This is supplemented by practice in converging both eyes on a dot which is brought closer and closer to the eye till it doubles. These combined exercises are a very considerable help in many cases.

5. The use of pilocarpine as a stimulant to the weak ciliary muscle has not seemed to me of much avail in these cases.

PARALYSIS OF ACCOMMODATION.

There is no hard and fast line between paralysis and insufficiency of accommodation. Any insufficiency of considerable amount may be called paresis and one that is complete or nearly so a paralysis. The terms paresis and paralysis are especially applied to marked insufficiencies of sudden development, and either of toxic origin or due to organic lesions of the nervous system. But this is a distinction hard to draw and maintain.

In the great majority of cases paralysis of accommodation is due either to poisons (cycloplegics) or to syphilis and its sequelæ (tabes, general paresis). Paralysis due to these conditions is too well known to require extended description. Of the syphilitic paralyses it may be said that they are late and very intractable manifestations, of evil prognosis, and yielding little or not at all to treatment, although they may disappear spontaneously and are then not infrequently replaced by paralysis of another kind. They are often unilateral and are usually, but not always, associated with paralysis of the sphincter iridis.

Other causes that we have seen are:

1. Non-syphilitic disease of the central nervous system (one case). Condition relieved by drainage of cisterna magna.

2. Traumatism (two cases; both associated with paretic mydriasis).

3. Disorder of metabolism in a growing boy (two cases; in one associated with almost complete paralysis of convergence).

4. Eyestrain produced by prolonged microscopic work with voluntary dissociation of the eyes (one case, associated with complete paralysis of convergence).

No diphtherial paralysis was seen, although one would infer from the text-books that diphtheria is a frequent cause of accommodative paralysis.

In toxic and traumatic paralyses the lesion is peripheral; in syphilitic and parasyphilitic paralyses it is either peripheral, basal, or nuclear. The other types of paralysis are central in origin and are, therefore, bilateral in character and are not associated with paretic mydriasis.

A very peculiar picture is afforded by a combined paralysis of accommodation and convergence. In two of my cases the paralysis of both functions was almost total and lasted for months, after which the conditions returned to normal. The symptoms were total inability to use the eyes for anything but distant vision, diplopia increasing *pari passu* as the object looked at approached the eyes, and severe pain in eyes, with in one case also marked headache.

The treatment of these conditions must necessarily be mainly causal.

UNEQUAL ACCOMMODATION.

We sometimes find the accommodation unequal in the two eyes.

Two reasons may be assigned for this inequality. Either the ciliary muscle may be weaker or the lens may be more rigid in one eye than in the other. The former sort of inequality occurs quite frequently in ophthalmoplegia interior due to syphilis, tabes, traumatism, or poisons. It can be readily understood that these conditions may affect one eye more than the other, or may affect one to the exclusion of the other. Since in these cases the sphincter iridis and the ciliary muscle are usually affected simultaneously, the inequality in accommodation is generally associated with inequality of the

pupils—the eye with the weaker ciliary muscle having the larger pupil.

When not due to the causes above mentioned unequal accommodation is usually not associated with inequality of the pupils. In this case, too, it may be caused by unequal action of the ciliary muscle. This was evidently the case in one of our patients, a man of thirty-one, in whom the accommodation in the right eye varied from 3 to 4 D, in the left from 5 to 6 D, and who also showed marked fatigue of the accommodation in the right eye. The causes here were obscure, but intestinal toxæmia and dental infection were present. The symptoms were great asthenopia and a marked spasmodic convergence when he tried to use his eyes for near work.

Most of the other cases of this sort seem to be due to an unequal rigidity of the lenses in the two eyes. In other words it is a form of presbyopia in which the sclerosis of the lens has advanced faster in one eye than in the other. Like simple premature presbyopia it is probably a mere physiological peculiarity producing no symptoms until the accommodation in one eye or the other has sunk so low as to require the addition of a glass for near work. When this occurs it is important to take account of the inequality and to determine by experiment whether we shall or shall not make an unequal addition for reading to the distance correction. Patients differ in this regard, some getting on better with such unequal addition, some without.

This condition of unequal presbyopia, or non-pathological inequality of accommodation, is found mainly in patients between the ages of 35 and 50.

ACCOMMODATIVE INERTIA.

Accommodative inertia is the condition in which the patient changes from one accommodative state to another sluggishly or with difficulty. It is analogous to the slow adaptation that some eyes show in passing from light to darkness or *vice versa*. It seems sometimes to be a source of real trouble. It is conceivably due to imperfect action of the ciliary muscle (ciliary inflexibility), and if so might be relieved by accommodative and particularly by converging exercises. But it seems more

likely that it indicates advancing sclerosis of the lens, in which case it would be a precursor to a fall in the accommodative power.

EXCESSIVE ACCOMMODATION.

Excessive accommodation like insufficiency of accommodation may be either ciliary or lenticular in origin.

A typical example of excessive accommodation due to ciliary overaction is the accommodative spasm produced by myotics, especially eserine. A similar, less violent action is set up naturally in some patients, causing them persistently to exaggerate a myopia or persistently to reject a hyperopic correction, in spite of continuous wearing. These cases of true spasm of accommodation may occur at any age up to 50 or even beyond. Such spasm may be associated with an actual weakness of accommodation, and in any case is not an instance of real excess of accommodative power.

Lenticular accommodative excess is the condition in which the accommodation is above the usual limit because the lens is less rigid than ordinary. It is the opposite of premature presbyopia and may be called delayed presbyopia. Whether, as would seem to be the case, it is always advantageous to have this overplus of accommodation, or whether the overplus may not sometimes indicate a pathological state,[1] our observations have not so far enabled us to determine.

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[1] In one case, there were a number of peripheral lenticular opacities, rather different in appearance from the ordinary arcus senilis lentis.

TENOTOMY OF THE INFERIOR OBLIQUE MUSCLE.[1]

By WM. CAMPBELL POSEY, M.D., Philadelphia, Pa.

(With six photographs on Text-Plate III.)

THE credit of proposing division of the inferior oblique muscle to correct certain ocular deviations belongs to Landolt, for, so far as I have been able to ascertain, the first mention of such a procedure is contained in a paper published by this author in the *Archives d'ophtalmologie* in 1885. After reviewing the anatomic relations and the physiologic action of the muscle, Landolt mentioned two conditions in which he thought division of its tendon indicated: (1) After paralysis of the superior oblique muscle, to counteract the loss in function of this muscle, the antagonist of the inferior oblique; (2) to prevent increase in myopia, by removing the pressure exerted by the inferior oblique muscle upon the venæ vorticosæ, thereby lessening the stasis in the veins of the uveal tract, which had been shown by Arlt to be a potent factor in the development of myopia. The communication was one of suggestion merely, and contains no mention of its trial by the author. Since its publication, Landolt has apparently given no other public expression to his views, and the literature contains no further mention of the subject of tenotomy of the inferior oblique until Duane again brought it to the attention of the profession in a comprehensive paper before the British Medical Association in 1906.

As Duane, with characteristic modesty, permitted this communication to remain unpublished, I shall, with his permission, record from his manuscript, which he has kindly

[1] Part of paper presented at the meeting of the American Ophthalmological Society, 1915.

placed at my disposal, the indications which he gave for tenotomy of the muscle in question.

INDICATIONS FOR THE OPERATION (DUANE).

The operation may be indicated in any deviation due either to actual overaction (spasm) of the inferior oblique, whether primary or secondary, or to a condition in which, without there being any actual involvement of the inferior oblique, the diplopia and symptoms are such as would be produced by spasm of that muscle. The following are the conditions for which tenotomy of the inferior oblique may be required:

I. *Conditions Simulating Spasm of the Inferior Oblique.*

A. Paralysis of the superior rectus of one eye. Fixation with the other eye. No true spasm.

II. *Conditions of True Spasm or Overaction of the Inferior Oblique Secondary to Paralysis of Other Muscles.*

B. Paralysis of the superior rectus of one eye. Fixation with the same eye, causing secondary deviation of the other eye due to spasm of its inferior oblique.

C. Paralysis of the superior oblique or some other depressor or elevator, with secondary spasm (contracture) of the inferior oblique in the same eye.

D. Paralysis of the abducens, with compensatory spasm of the inferior oblique in the same eye.

III. *Primary Spasm of the Inferior Oblique.*

E. Spasm of the inferior oblique not due to paralysis of other muscles in the same or in the other eye.

CONSIDERATION OF THE INDICATING CONDITIONS. VARIETIES OF SPASM AND PSEUDOSPASM OF THE INFERIOR OBLIQUE.

A. *Paralysis of the Superior Rectus without Spasm of the Inferior Oblique of the Other Eye.*

These are cases in which the unparalyzed eye is used for fixation. Here we operate according to the principle of

ILLUSTRATING DR. POSEY'S ARTICLE ON "TENOTOMY OF THE INFERIOR OBLIQUE MUSCLE"

A

B

C

D

FIG. 1.—Case 3.—Insufficiency of L. S. R., with overaction of R. I. O. A. Primary position; eyes straight. B. Looking slightly to right; O. S. lags, O. D. fixing. C. Looking to left; O. D. tilts up and in. D. Looking to left after operation; O. D. up and in, tilt much less pronounced.

Fig. 2.

Fig. 3

Case 9.—Paresis of both superior and right rectus muscles.

FIG. 2.—Shows torticollis; head held in oblique position, right side of face presenting. Gaze is directed to the extreme left; O. S. is fixing and O. D. deviated up and in.

FIG. 3.—Shows almost perfect parallelism of visual axes (O. D. slightly divergent) after tenotomy of both inferior obliques and advancement of right rectus externus.

Graefe that we can most satisfactorily compensate for the deficiency of movement in a paralyzed eye by operating on the associate in the sound eye, thus producing in the latter an artificial paralysis which weakens it in the same sense and for the same directions of the gaze that the natural paralysis weakens its fellow.

Why not advance the superior rectus in these cases? Often, of course, this would be the operation of election, but in some cases advancement is inapplicable because we are dealing with absence of nerve power. Here the operation would simply draw the eye up mechanically without helping it to move up in any normal manner. Again, in some congenital cases we may find, when we start to do an advancement, that the muscle itself is wanting or is replaced by a fibrous cord. Here, of course, advancement is out of the question and might even do harm.

Another objection against relying upon advancement in cases of marked superior rectus paralysis is that a very thoroughgoing advancement produces marked restriction of movement in the field of action of the antagonist; *i. e.*, in this case would produce in the lower field a diplopia which may be very annoying. Moreover, in such extensive advancements we can seldom regulate the effect with the proper accuracy, and the final result may differ in many degrees from the immediate one. Hence, in a case of very extensive deviation we shall get a better result if we first remove a large part of the deviation by a tenotomy of the contralateral inferior oblique, then correct the rest by an advancement, which, being only moderate in extent, will not hamper unduly the action of the opposing depressor and can be more readily regulated and controlled than if very extensive in amount.

B. *True Spasm of Inferior Oblique Secondary to Paralysis of the Superior Rectus in the Other Eye.*

This occurs when the eye with the paretic rectus is habitually used for fixation. In such a case the sound eye goes into a position of marked secondary deviation from overaction of the associate to the paralyzed muscle, the associate here being the inferior oblique.

This sort of secondary deviation occurs mainly when the paralysis is incomplete, often quite trivial. Fixation with

the affected eye is frequent in superior rectus paralysis, especially when the latter is traumatic or congenital, and may even occur when the eye used for fixation is the poorer-sighted of the two. Slight congenital paralysis of this sort, with secondary deviation of the other eye, is quite a frequent condition. I have seen a number of such cases.

Referring to a case, the notes of which are appended, Duane goes on to say:

This case is a typical specimen of a group, the symptoms of which, constituting a fairly well-marked syndrome, may be stated as follows: In a case of right-eye paresis with secondary left-eye spasm we find, if the left eye is covered, a—(1) Moderate or often only a very slight restriction of the movement of the right eye up and to the right. (2) With both eyes open, a marked upshoot of the left eye when the eyes are directed up and to the right. (3) Vertical diplopia with the image of the left eye below, this diplopia increasing fast as the eyes are carried up and to the right. (4) Tilting of the head toward one shoulder, usually the right. This is evidently done to obviate the diplopia. The attitude may be maintained so persistently as to cause an actual wry-neck. (5) The condition is not only congenital, but in some cases also appears to be hereditary.

If, as in the case cited, the disfigurement produced by the oblique movement is marked, and the diplopia is large in amount and causes some embarrassment of vision, and if there is a persistent wry-neck, tenotomy of the inferior oblique is indicated and would certainly be of service.

C. *Spasm of Inferior Oblique Secondary to a Paralysis of a Depressor or Elevator (Especially the Superior Oblique) in the Same Eye.*

In a case of this sort tenotomy of the inferior oblique would be resorted to only if (a) we were convinced that the condition is going to be permanent and stationary; (b) that the symptoms are such as to justify the operation.

D. *Primary Spasm of the Inferior Oblique.*

These cases are probably very rare. They should have one feature in common with the cases classed under category C, which distinguishes them from those classed under A. In a spasm of the right inferior oblique due to a paresis of the

left superior rectus, the left eye will fly up when turning to the left to make a parallel movement with the right eye, but it will not fly up when turning to the left to converge with its fellow, because in this action the left superior rectus is not specially called into play, at least as an elevator. In a spasm of the right inferior oblique, either primary or due to paralysis of a muscle in the right eye, the eye will fly up when turned to the left to perform either a convergent or a parallel movement. In such a case we may find binocular vision for distance combined with a marked vertical squint for near.

SUMMARY.

Tenotomy of the inferior oblique is indicated in:

A. Complete stationary paralysis of the superior rectus of the other eye, especially when this paralysis is traumatic or congenital.

B. Partial paralysis or even slight insufficiency of the superior rectus of the other eye when, owing to fixation being performed by the latter, the fellow develops a spasm of the inferior oblique so pronounced as to give rise to a disturbing (even if unnoticed) diplopia, a disfiguring upshoot of the affected eye, or a lasting torticollis. Such an indication is furnished especially by congenital cases.

C. In spasm of the inferior oblique, either secondary to paralysis of the superior oblique or some other muscle in the same eye, or else occurring as a primary affection, provided in either case the deviation and diplopia produced by the spasm are sufficiently disturbing to warrant an operation, and provided also that the condition gives good evidence of being stationary.

Having had occasion about the time Dr. Duane was engaged in the preparation of this communication to consult him about some details of a paper which I was myself preparing on the subject of congenital squint, and which was read before the Section on Ophthalmology of the American Medical Association in 1907, I first learned from him of the operation upon the inferior oblique and of the conditions in which he deemed the procedure justifiable. I immediately began to

perform the operation, and in the paper just cited gave the notes of five cases which had been subjected to it. Since that time I have performed tenotomy of the inferior oblique upon twelve other cases. From these seventeen cases, with those of four others kindly placed at my disposal by my colleague, Dr. Zentmayer, I have selected the following for a detailed description:

CASE 1.—Female, aged twenty-six years, stenographer. For years past supraorbital headache, increased by near work. Left eye always the weaker. Add. good. R. H. 4°; esophoria 4°. Diplopia for far in connection with headache, associated with nausea. Pain more apt to be localized over O. S. Following correction prescribed and worn with comfort for three years: O. D. sph. − 1 D. ⊂ Pr. 1½° base down = $\frac{5}{6}$; O. S. sph. − 0.75 D. ⊂ cyl. − 0.25 D. axis 10° ⊂Pr. 1½° base up = $\frac{5}{6}$. Examination showed marked overaction of R. I. O. when object carried to left. All other movements good except limitation of upward and outward motion in O. S.

Diagnosis.—Congenital insufficiency of L. S. R. muscle with overaction of R. I. O.

Tenotomy of R. I. O. Immediately afterward, both eyes on same horizontal plane when looking to left. Now R. H. ½; exophoria ½. Orthophoria at near. Two months later orthophoria at all distances. No uptilting in O. D. No diplopia. Headache cured. Not seen since.

CASE 2.—Female, aged thirty-four years. Formerly a stenographer. First saw double seven years before. Since then double vision has persisted with pain in O. D. Has worn for twelve years O. D. sph. + 0.37 D. ⊂ cyl. − 1.25 D. axis 10° ⊂ Pr. 4° base down = $\frac{5}{6}$; O. S. cyl. − 1 D. axis 170° ⊂ Pr. 1½° base up = $\frac{5}{6}$. With this correction, after close work, lines begin to slant, and diplopia occurs in the upper left field. Examination revealed some impairment of upward rotation in O.S. and up- and in-shoot of O.D. when objects directed to the left. R. H. 6°; esophoria 1°.

Diagnosis.—Probable congenital insufficiency L. S. R. with overaction R. I. O.

Tenotomy R. I. O. Eyes now on same horizontal plane. Diplopia has disappeared. Pain in O. D. relieved, but R. H. 5°; exophoria 1½°. At end of four months, R. H. 5°; vertical orthophoria. F. O. D. sph. − 1.25 D. ⊂ cyl. + 1.50 D. axis 90° ⊂Pr. 2° base down; O. S. sph. − 1.75 D. ⊂ cyl. +2.25 D. axis 75° ⊂ Pr. 2° base up. Recurrence of pain. Advancement L. S. R. advised but refused.

CASE 3.—J. E. B., aged thirty-five years, casket maker, states that O. D. has been "crooked" when he looks to the left since birth. Frequently sees double for near, which interferes greatly with his work. Occasional dizziness. Claims vision good, both for near and far. No family history of strabismus. In primary position, eyes straight. Movements normal to the right. To the left, along the horizontal plane, O. S. fixes, O. D. tilts up and in. On upward motion, O. S. lags, particularly in motions up and out (Fig. 1). V. O. D. cyl. + 0.25 D. axis 145° = $\frac{5}{5}$; O. S. cyl. + 0.50 D. axis 115° = $\frac{5}{5}$. R. H. 18°; exophoria 2°. (Fig. 1.)

Diagnosis.—Insufficiency of L. S. R., with overaction of R. I. O.

Tenotomy R. I. O., reducing up and in tilt of O. D. One week after operation, R. H. 8½°; exophoria 2°. Not seen since.

CASE 9.—Male, aged ten years. (Previously reported, Case 6.) Good mentality; head well formed, but face underdeveloped; pronounced wry-neck; head held in oblique position, right side of face presenting. Dr. James K. Young reported contraction of left sternocleidomastoid muscle, with chin toward the right; left cervical, right dorsal, and slight left lumbar curve; slight hemiatrophy of left side of face. Primary position O. D., convergent strabismus, 45 degrees; object carried to the right, O. D. fixing, O. S. deviates up and in; object carried to the left, O. S. fixes and O. D. deviates up and in; both eyes deviate upward under cover; marked limitation in outward rotation of O. D. (Figs. 2 and 3).

Diagnosis.—Insufficiency of both S. R. and R. E. R. muscles.

Tenotomy of both I. O. muscles. Removed the upward and inward deviation of the eyes. Advancement of R. E. R. resulted in parallelism of visual axes; two months after operation no appreciable change in wry-neck.

CASE 10.—Wills Hospital. Female, aged five years. Eyes crossed since six months of age, O. D. mostly, O. S. at times. No family history of strabismus. On examination, O. S. fixes, O. D. convergent 35° and turned slightly up. When O. D. made to fix, O. S. assumes similar converging position. Horizontal plane, movements to right, O. D. fixes and O. S. follows, but goes up and in at extremity of inward rotation. O. D. outward rotation good. Movements to left, O. S. fixes. Rotates externally normally. O. D. rotates up and in. Upward rotation of both eyes defective.

Diagnosis.—Congenital paresis of both S. R. muscles.

Under atropin, V. O. D. sph. + 2 D. = $\frac{5}{9}$; O. S. sph. + 2 D. = $\frac{5}{7.5}$.

Division of both I. O. muscles under ether. Immediate relief to up and in tilt in lateral deviations in each eye. Residual alternating convergent squint of 20°. Correcting glasses prescribed. Six months later, eyes almost straight with glasses. Alternating convergent squint of 20° when glasses are removed. Not seen since.

CASE 15.—Wills Hospital. Male, aged twenty-eight years. O. D. convergent since three years of age, following convulsions. Has been wearing high M. + Am. lenses since seven years of age. On examination, O. D. convergent 35° and turned slightly up. Outward rotation in O. D. limited, also in O. S. Upward motion in O. S. limited, especially up and out.

Diagnosis.—Insufficiency of both E. R. muscles and of L. S. R. muscle. Overaction of R. I. O. V. O. D. = $\frac{1}{60}$, not improved by high minus lens. Lens partially cataractous; O. S. = $\frac{3}{60}$ sph. − 9 D. ⊂ cyl. − 1 D. axis 170° = $\frac{5}{7.5}$.

Tenotomy of R. I. O. and advancement of R. E. R. One month later, O. D. still convergent 15°, but upward deviation has gone. Two months later, O. D. convergent 25°, but eyes on same horizontal plane.

CASE 16.—Wills Hospital. Boy, aged five years, said to have squinted since infancy. Several members on maternal side said to have had similar squint. Not verified. On examination, O. D. fixes, O. S. convergent 40°. Head abnormally large. Orbits appear shallow, oblique palpebral fissures. Horizontal plane, object carried to the left, O. D. fixes, O. S. turns down and out. Object carried to right, O. D. fixes, O. S. goes up and in. In upward motion, O. S. lags, especially out and up. In downward motion, O. S. deviates down and in. Monocular fixation O. S. Inward motion with O. S. good. Up and in poor, down and in good, outer good. Movements of eye not influenced by movements of jaw. Drs. Schwenk and Zentmayer concurred in diagnosis of paralysis of L. S. R. and overaction of L. I. O. in conjugate motions to the right. V. O. D. sph. + 1 D. = $\frac{2}{6}$; O. S. sph. + 1 D. = fingers at 1 meter.

Diagnosis.—Insufficiency of L. S. R. muscle with overaction of L. I. O. in conjugate motion to right.

L. I. O. divided. Upward and inward shoot of O. S. when object carried to the right is somewhat lessened. Eye drops down and slightly out as before in ordinary fixation. Object carried to the left, O. S. drops down and out. Six months later, in medium line, eyes straight. Object carried to the

left, O. D. fixing, O. S. goes down and out. Advancement of S. R. performed under ether, bringing eye upon same horizontal plane as O. D. O. S. still convergent 20°. Not seen after glasses prescribed.

CASE 21.—Male, aged forty years. Six years ago diplopia with drooping of lids following eruption on body. Vision also began to fail progressively. Antisyphilitic treatment inaugurated. At time of first examination by me ocular palsies were said to have remained unchanged during past four or five years.

Fixes with O. D., O. S. being turned markedly up and in. When made to fix with O. S., O. D. turns down and slightly out. Pronounced double ptosis. In O. S. paresis of external inferior superior and internal rectus muscles. Obliques alone seem conserved. In O. D. all muscles paretic, with exception of external and inferior rectus muscles. Vision reduced in O. D. to $\frac{3}{4\frac{8}{8}}$, in O. S. to $\frac{4}{6\frac{0}{0}}$, in consequence of simple atrophy of optic nerves.

Diagnosis.—Ophthalmoplegia externa with optic atrophy in consequence of cerebrospinal syphilis. Overaction of L. I. O. in consequence of paralysis of R. S. R. muscle.

Tenotomy of L. I. O. and L. S. R., reducing up and in deviation of O. S. at least one-half. One month later, eyes on same horizontal plane, and head, which had been held in oblique posture, now almost straight.

As indicated in the notes of the cases, all, with the exception of Case No. 21, were congenital.

The most common type is that illustrated by Cases 1, 2, 3, 4, 5, 6, 7, 8, 16, 18, and 19, and consisted of insufficiency of the superior rectus of one eye and overaction of the inferior oblique of its fellow. (Class II—B, Duane.)

The next most frequent type was insufficiency of both superior rectus muscles, with overaction of both inferior obliques, as illustrated by Cases 9, 10, 11.

Insufficiency of the superior oblique with spasm of inferior oblique of same eye (Class II—C, Duane) is illustrated by Case 12.

The remaining cases are of mixed types, as follows:

Case 13.—Insufficiency of L. E. R. and L. S. O. Overaction of L. I. O.

Case 14.—Insufficiency of R. E. R. and L. S. R. Overaction of R. I. O.

Case 15.—Insufficiency of both E. R. and of L. S. R. muscles. Overaction of R. I. O.

Case 17.—Insufficiency of L. S. R. Overaction R. I. and R. I. O. muscles.

Case 20.—Insufficiency of L. S. R. Overaction R. and L. I. O.

Case 21.—Acquired external ophthalmoplegia, with overaction L. I. O., in consequence of paralysis of R. S. R. muscle.

The technic of the operation performed by Duane, and which I have followed, is the same as that suggested by Landolt, and consists in a division of the tendon of the muscle at its origin in the lower-inner portion of the orbit. After injecting the tissues with a 2 per cent. solution of novocain, a curvilinear incision is made just superior and parallel to the lower and inner bony rim of the orbit, the skin and sublying tissues being divided with a scalpel down to the bone. A strabismus hook is now inserted into the wound and gentle traction made upon the tissues until the muscle is encountered. This is assured by the upward motion imparted to the globe by traction on the hook. The tendon of the muscle is then drawn forward into the wound and divided by a few snips of Stevens scissors. One or more stitches are inserted to close the wound, and a firm dressing applied. This latter is removed at the end of forty-eight hours, when the lips of the wound will be found to be firmly united, the only evidence of the traumatism of the operation being in some cases a subconjunctival ecchymosis of the lower-inner part of the globe, which disappears after a few days.

The results of the tenotomy were all more or less satisfactory. The up and in tilt imparted to the eye by the overaction of the muscle generally disappeared, and in a number of the cases immediate relief was experienced from the traction which the muscle had exerted upon the globe and from an annoying diplopia.

In the cases in which it was possible to ascertain the hyperphoria, prismatic measurement indicated that the tenotomy had reduced the deviation in amounts varying from 3 to 17 degrees. The lateral balance of the muscles was also improved, no doubt in consequence of the greater power given the adductors and abductors by the lessening of the vertical

deviation. In none of the cases did any scarring of the lid follow the operation, nor did it produce any gross ocular deviation or interfere in any way with the proper action of the muscles that had not been disturbed.

I believe the operation a procedure of great value to meet the indications outlined by Duane, especially, however, in relieving the up and in tilt imparted to the eye by the over-action of the muscle in conjugate lateral movements where the superior rectus muscle of the fellow-eye is paretic.

In my paper on congenital squint I questioned the desirability of any form of operative procedure upon the eyes of children who had squinted from birth, until the age of at least ten years had been attained, as the permanent character of the ocular deviation is rarely fully established until that age. I now believe that it is wise to wait in some instances even longer, and to defer operation until five or six years later, as I have found in some of my cases, upon whom no operation was performed, that the development of the orbits, conjoined with the careful correction of the refraction error, which is often not inconsiderable in this class of cases, will at times render the muscular deviation negligible and the necessity for operative measures may be avoided.

As both eyes are employed in fixation and there is no danger of the development of amblyopia ex anopsia, the necessity for early refraction is not as urgent in congenital as in concomitant squint. If there is considerable ametropia, however, I usually prescribe the correcting lenses, as determined by the retinoscope, at about three years of age. Orthoptic exercises are rarely of service.

ROSACEA KERATITIS AND CERTAIN OTHER FORMS OF MARGINAL KERATITIS, NEUROPATHIC IN ORIGIN. TREATMENT BY PERICORNEAL NEUROTOMY.[1]

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AS is well known, there sometimes occur associated with herpes facialis lesions in the central portion of the cornea. The most common etiologic factors in their occurrence seem to be acute coryza and gastro-intestinal disturbances due to improper food, but often no definite cause is apparent, owing, no doubt, to its trivial nature. These corneal lesions I have discussed somewhat at length elsewhere.[2] Not generally known is the fact that under the same conditions there may occur lesions of highly distinctive character limited to the periphery of the cornea.

Since it may now be accepted that facial herpes is neuropathic in origin and due to some irritative condition in the Gasserian ganglion,[3] it is altogether probable that these peripheral corneal lesions are likewise so. But there is another strong reason for believing them to be neuropathic; namely, their similarity to the central lesions of corneal herpes. In

[1] Read at meeting of American Ophthalmological Society, 1915.

[2] *Jour. A. M. A.*, 1909, liii., p. 191. Archives of Ophth., 1911, xl., p. 486.

[3] Pneumococci have been found in the Gasserian ganglion in cases of herpes, and recently Rosenow and Oftendal (*Jour. A. M. A.*, June 12, 1915, p. 1968) claim to have experimentally produced herpes of the skin in animals by intravenous injections of special strains of streptococci. The skin lesions did not contain the organisms, but the corresponding ganglia showed hemorrhagic lesions from which the organisms were recovered.

fact, they differ from the latter only in their situation and arrangement. They usually first appear as round, oval, or slightly irregular infiltrates immediately beneath the surface, and vary in size from a fraction of a millimeter up to several millimeters in diameter. According to the severity of the process the epithelium may or may not remain intact over them, and they may or may not become slightly elevated, but they seldom undergo ulceration. They are stained by fluorescin in the same characteristic way as neuropathic lesions in general. As a rule, they are situated at a distance of about 1½*mm* from the limbus, and when multiple, often occur in a row running parallel with the limbus. Careful examination in the early stages shows that the larger spots are really made up of smaller ones, just as are the lines and spots in dendritic keratitis. This is most easily determined by staining them with fluorescin and after washing away the stain from the conjunctival sac examining them quickly before the color has time to diffuse. The conjunctival congestion, which is most marked in the vicinity of the lesions, as well as the subjective symptoms of irritation, may be considerable or insignificant. Sometimes there are associated with the corneal lesions the herpetic nodules at the limbus described by me elsewhere. The duration of the process varies from a few days to a number of weeks. Vascularization of the cornea seldom occurs. In rare cases the lesions consist of vesicles from which filaments may sometimes grow.

Assuming the lesions to be neuropathic, their usual location at an approximately uniform distance of 1½*mm* from the limbus plainly shows, it seems to me, that they occur at the terminations of the conjunctival nerves in the cornea. For it is known that the periphery of the cornea is supplied in part by nerve filaments which extend into it from the conjunctiva. I have never been able to determine any reduction in the sensibility of the periphery of the cornea in these cases, but have attributed this to the fact that this portion of the cornea is normally rather insensitive and that it is partly supplied also by the true corneal nerves. In exceptional cases, in addition to the lesions near the limbus, I have observed one or two spots as far as 3 or 4*mm* from the limbus. These I have explained by assuming that occasionally some of the conjunctival

nerves extend unusually far into the cornea, and this assumption seems to be fully confirmed by the results of the operative treatment to be described.

The occurrence of these corneal lesions at the terminations of the conjunctival nerves is perhaps simply a manifestation of the general tendency of herpetic lesions to occur at the periphery of an area of nerve distribution. Thus, facial herpes is most apt to involve the lips or alæ of the nose. In any case it is necessary to assume that the particular ganglion cells or nerves corresponding to these regions are especially vulnerable. Elsewhere I have suggested an explanation of the lesions of neuropathic keratitis in general, and this applies equally well to the marginal form under consideration. It is, briefly, that impulses from the affected ganglion cells pass backward along the ordinary sensory nerves to the nerve terminals in the cornea, where they produce, by electrolytic dissociation, toxic substances injurious to the tissues. I have also described the histology of the lesions of superficial punctate keratitis which undoubtedly are essentially the same as those under consideration, and have shown them to consist of localized infiltrates of necrotic pus-cells situated immediately beneath Bowman's membrane.

In cases of rosacea of the skin, as is well known, there are sometimes marginal infiltrates or ulcers of the cornea.[1] In long-standing and severe cases these may repeatedly occur, and the cornea become superficially vascularized so that a more or less extensive pannus-like condition is finally produced. The affected eye is usually extremely irritable, but the visual acuity is reduced only in the cases of pannus formation, usually the alcoholic cases. Fortunately, the lesions seldom become seriously infected with pyogenic organisms.

For several years I have regarded and treated rosacea keratitis as a form of neuropathic keratitis. My reason for this has been that careful examination of the lesions has convinced me that they are identical in nature with those of marginal neuropathic keratitis just described. Their situation is the same, their sizes, shapes, arrangement, and staining

[1] In my experience rosacea keratitis is far more frequent than the rosacea conjunctivitis of Arlt, in which there are phlyctenule-like nodules at the limbus.

reaction with fluorescin are the same. The only differences are the longer duration and consequently greater tendency to vascularization of the rosacea lesions. These are differences of degree, not of kind, and may be attributed to differences in the agents producing the nerve lesions.

In long-standing cases of rosacea, especially the alcoholic cases with vascularization, the lesions may occur far out in the cornea. A possible explanation for this is that, owing to the long duration and frequent recurrence of the process, nerve filaments from the conjunctival branches grow out abnormally far into the cornea.

If the foregoing conclusions are correct, and the corneal conditions described are really due to impulses passing along the conjunctival nerves, the lesions should promptly heal if the abnormal impulses are prevented from reaching the cornea. A simple method of cutting off the impulses would be to incise the conjunctiva in the region involved, that is, to do a partial peritomy. Within the past year I have done this in fifteen consecutive cases with most successful and conclusive results. In every case healing promptly occurred. In twelve cases the corneal lesions failed to stain with fluorescin at the end of forty-eight hours. In the others they could be stained slightly for a day or two longer. These results seem to prove conclusively not only that rosacea keratitis and the other forms of marginal keratitis described are neuropathic in origin, but that they are due to impulses reaching the cornea through the conjunctival nerves.

Four of the cases operated upon were cases of rosacea keratitis, in two of which there was vascularization. In one of the latter there had been recurring attacks for two years; in the other rosacea cases the durations were each about one month. The age of the patient in three rosacea cases was about fifty-three years, and in the other twenty years. The other cases were of the simple herpetic type. In three cases there was acute coryza and in two others herpes facialis. In the remaining cases the diagnosis was based chiefly upon the appearances of the corneal lesions alone, but was substantiated by the results of the operative treatment. In one of the herpetic cases the age of the patient was fifty-six years; in the others the ages were from seventeen to thirty-three years. The

durations of the cases before operation as given by the patients were from three days to two months. In one case the lesions consisted of four epithelial filaments in a row attached to the cornea at a distance of 1¼*mm* from the limbus. Forty-eight hours after operation these had completely disappeared. In the other cases there were definite infiltrates, and these, of course, remained visible for some time after they could no longer be stained. The subjective symptoms of irritation promptly subsided in all but two cases. In one of these they persisted for twelve days in spite of the disappearance of the corneal lesions. In the other case the peripheral lesions healed promptly, but a dendritic figure appeared near the center of the cornea which healed in about a month under holocain treatment. This probably was due to a coincident involvement of the true corneal nerves.

The method of performing the operation that was used in most of the cases, and that I now employ, is as follows: The conjunctiva is incised at the limbus by means of scissors for a considerable distance beyond the limits of the corneal lesions, care being taken to cut all the tissue down to the sclera. In some cases I have extended the incision over more than one-half the circumference of the cornea. The conjunctival flap is then undermined by snips of the scissors for a distance of 4 or 5*mm* back of the limbus so as to sever as far as possible all nervous communications. The flap is then brought up to the corneal margin by means of two sutures, one at each end of the incision, as in the Van Lint operation for cataract. In the first few cases I also cut into the sclera at the limbus with a cataract knife, but later found this unnecessary. In one case I curved the conjunctival incision a considerable distance from the limbus and undermined the flap remaining. The result, so far as the corneal lesions were concerned, was perfectly satisfactory, but there was more conjunctival reaction than in the other cases. Perhaps this modification, however, should be given a further trial. Since the purpose of the operation is different from that of peritomy, and the technic is also somewhat different, it may be well to distinguish it by the term "pericorneal neurotomy."

Whether or not the results of the operation will in all cases be permanent I am not yet prepared to say. None of my

patients, however, has returned with a recurrence. It would seem probable that the severed nerves would regenerate, but their new distribution might, as a rule, be so widespread that no corneal lesions would result. It is obvious that the same condition might later occur in another part of the cornea or even in the same part, due to other cells of the ganglion becoming affected. The operation is so simple and free from danger, however, that there is no reason why it should not be repeated as often as indicated.

Rosacea of the skin is generally believed to be an angioneurotic condition due to some abnormal constituent of the blood. The reason for this belief is that the condition seems to be primarily a dilatation of the vessels of the skin in certain regions. The acne lesions sometimes associated with it are now believed to be secondary and not essential to the process. Whether the affected vessels are dilated by stimulation or depression of the vasomotor nerves or the ganglion cells does not seem to have been discussed. The question as to what the abnormal constituents of the blood are that cause rosacea apparently also has not been discussed. It is believed that improper diet is the most common cause, and we know that chronic alcoholism often gives rise to the condition. Judging from my own cases, tea drinking is a common cause. Possibly rosacea is not due to the presence of alcohol, tea, etc., in the blood, but is indirectly due to the chronic intestinal disturbances set up by them. As a result of the latter, incompletely digested proteins or other substances could be absorbed in abnormal quantities and produce deleterious effects on the nerves or ganglion cells. That transient gastrointestinal disturbances, although relatively slight, can affect the Gasserian ganglion is indicated by the experience of any one repeatedly subject to "cold sores."

Having shown, conclusively it seems to me, that the corneal lesions of rosacea are neuropathic, and assuming the skin lesions to be angioneurotic in origin, how may they be correlated? The corneal lesions may readily be explained by assuming that the same deleterious agent in the blood that produces the skin lesions, from time to time, acts injuriously on the Gasserian ganglion. In regard to the way in which this deleterious agent produces the skin lesions a number of possi-

bilities suggest themselves. It might do so by acting upon the vasomotor endings, on the vasomotor centers, or upon the Gasserian ganglion. In the latter case the vasodilatation could be produced either reflexly by afferent impulses from the ganglion to the brain, or by efferent impulses from the ganglion to the skin. The latter impulses could cause dilatation of the vessels either indirectly, by producing irritating substances at the terminals of the sensory nerves, or directly through the vasomotor nerves. This last possibility is supported by the recent experimental observations of Bruce,[1] which go to show that the nerves from the posterior root ganglia give off vasomotor branches at the periphery.

Obviously, the problem as to the explanation of the skin lesions of rosacea is a complicated one, difficult of exact solution. Since, however, the cornea has no vasomotor nerves, and the Gasserian ganglion must therefore be responsible for rosacea keratitis, the most reasonable assumption at present would seem to be that the skin lesions of rosacea are likewise due to irritation of certain cells in this ganglion.

[1] A. N. Bruce: *Quarterly Journal of Exp. Phys.*, 1913, vi., p. 339.

BACTERIOLOGICAL AND CLINICAL STUDIES OF AN EPIDEMIC OF KOCH-WEEKS BACILLUS CONJUNCTIVITIS ASSOCIATED WITH CELL INCLUSION CONJUNCTIVITIS.[1]

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(With 55 figures on Text-Plates IV., V., VI.)

WHILE engaged in the study of conjunctivitis associated with epithelial cell inclusions, the occurrence of an epidemic[2] of conjunctivitis due to hæmoglobinophilic bacilli enabled us to investigate the relationship between this organism and the epithelial cell inclusions.

Since the discovery of the inclusions by von Prowazek and Halberstaedter, who considered them to be the ætiological agents of trachoma, an abundant literature has arisen. Opinions are still greatly divided as to the nature of the bodies, although the majority of investigators regard them as an independent organism, as claimed by von Prowazek and Halberstaedter. They do not, however, commit themselves to the statement as to whether the organism represents the causative agent of trachoma or not. We were inclined to the belief that it constitutes an independent organism which produces a true conjunctivitis of a more or less characteristic clinical course. Herzog[3] considered them to be mutation

[1] Read before the Ophthalmological Section of the New York Academy of Medicine, May 17, 1915.

[2] Service of Dr. Martin Cohen at the Randall's Island Hospital, New York.

[3] Herzog, H., *Arch. f. Ophth.*, 1910, lxxiv., 520.

forms of the gonococcus, while Williams[1] views them as the cell inclusions of various organisms, such as the hæmoglobinophilic bacillus and allied species.

Although by means of a special method an organism strikingly similar to the epithelial cell inclusions has been made to grow in pure culture,[2] their pathogenic properties, nevertheless, have not yet been established.

As a result of our previous studies a tentative conclusion had been reached that infection of the conjunctiva with these organisms produces an independent conjunctival disease which may properly be described as cell inclusion conjunctivitis. This organism may be present alone, or it may be associated with other pathogenic organisms.

OUTBREAK AND COURSE OF THE PRESENT EPIDEMIC OF KOCH-WEEKS BACILLUS CONJUNCTIVITIS

Prior to the outbreak of the Koch-Weeks bacillus epidemic,[3] one of us (Cohen) had under observation for about five months ten cases of inclusion conjunctivitis and four cases at the outdoor department of the Post-Graduate Hospital. In these cases, smears (and in a few cases, cultures) were taken weekly and examined (Noguchi), but no Koch-Weeks bacilli were found. On October 3, 1914, one of the inclusion cases under observation at the Island developed an acute conjunctival inflammation. In the conjunctival smears numerous Koch-Weeks bacilli, as well as epithelial inclusions, could be demonstrated. The acute conjunctival inflammation spread to the remaining nine inclusion cases. The smears from all showed inclusions as well as numerous Koch-Weeks bacilli.

In an adjoining ward there were under treatment fifteen cases of follicular conjunctivitis, three cases of interstitial

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[1] Williams, A. W., *Collected Studies from the Bureau of Laboratories, Department of Health, City of New York*, 1912–13, vii., 159–247; *Jour. Infect. Dis.*, 1914, xiv., 261.

[2] Noguchi, H., and Cohen, M., ARCH. OF OPHTH., 1914, xliii., 117; *Jour. Exper. Med.*, 1913, xviii., 572.

[3] In the present article the hæmoglobinophilic bacilli found in these cases were designated as Koch-Weeks bacilli, in order to conform with the usage of this term in ophthalmology and bacteriology when speaking of this class of organisms.

keratitis, and one case of chronic dacryocystitis. Thirteen of these contracted the Koch-Weeks bacillus infection. During the entire course, which lasted from two weeks to seven months, no pathogenic organisms but the Koch-Weeks bacilli could be demonstrated in the smears and cultures.

There were seven patients in the trachoma ward. Two of these showed simultaneous presence in the conjunctiva of inclusion and of Koch-Weeks bacilli. The latter organisms were present only in cultures, as there were possibly too few to be recognized in the smears. This condition was present for a short time during this epidemic. The infection with the Koch-Weeks bacillus apparently produced no change in the clinical picture of these trachoma cases. The four inclusion cases not treated on the Island showed at no time during the past year the Koch-Weeks bacillus, either in smears or cultures; not even during a relapse or reinfection, of which one of these cases had two, could they be demonstrated. Table I. shows the bacteriological findings in the cases on which the present report is based.

TABLE I.

Organisms present.	*No. of cases.*
1. Epithelial cell inclusions alone	6
2. Epithelial cell inclusions at first, with subsequent Koch-Weeks infection	17
3. Koch-Weeks bacillus alone	13
4. Koch-Weeks bacillus at first, with subsequent appearance of inclusions	2
5. Pneumococcus with inclusions	1
Total	39

In acute catarrhal conjunctivitis it is at times difficult, or impossible, to determine the causative oganism from the clinical manifestations alone. This is due to the great variability in virulence of the usual infecting organisms, and to the resisting qualities of the individual patient. Yet in many of the acute infections of the conjunctiva, the ætiological diagnosis as made by the clinical appearance could be confirmed subsequently by the bacteriological diagnosis, especially in the subacute state. In mixed infections, such a diagnosis is much more difficult.

In a few instances, even smears and cultures of the conjunctival scrapings fail to clear up the diagnosis, as no pathogenic organisms can be found. That the microscope alone is not sufficient to exclude the presence of an organism is shown by the fact that the scrapings from cases of trachoma which showed neither inclusions nor the Koch-Weeks bacilli (as proved similarly by cultures), when inoculated on the conjunctiva of the higher apes (baboons), produced symptoms similar to those which are seen in inclusion cases in man. They were, however, of a milder type and lasted only about ten days. The conjunctival scrapings from the inoculated animals revealed the presence of epithelial inclusions, but no Koch-Weeks bacilli or allied organisms appeared in the smears or in the cultures.

We shall now discuss the clinical manifestations in the group of cases mentioned in Table I.

The clinical course of inclusion conjunctivitis has already been described by Cohen.[1] In the early stage, the clinical manifestations resemble those seen in the Koch-Weeks bacillus infection, and it is only by the examination of smears or cultures that the diagnosis can be positively made. Briefly, they are as follows: In the beginning there is moderate œdema of the lids, with mucopurulent secretion. Somewhat later isolated reddish translucent follicles appear in the lower palpebral conjunctiva and upper folds, these two sites becoming involved simultaneously. At a later stage, the upper palpebral conjunctiva takes on a brick red color and assumes the characteristic granular appearance. These regular and progressive manifestations of the disease retrogress after about two months by absorption of the contents of the follicles and papules. These disappear first from the upper portions of the conjunctiva, when the conditions resemble those of cases of follicular conjunctivitis; and later, in about three months, they begin to disappear from the lower conjunctiva. At the end of four months, the conjunctiva is again in a normal condition.

This is the usual course, but at times there are deviations, due to external or unknown conditions, or to a previous affec-

[1] Cohen, M., ARCH. OF OPHTH., 1913, xlii., 29.

tion of the conjunctiva; as, when grafted on a follicular conjunctivitis, the original follicles become larger and new ones make their appearance. In the stage of retrogression the papules become absorbed and the original follicles, owing to relaxation of surrounding pressure, again become prominent, the course being then one of a follicular conjunctivitis. In two cases of inclusion conjunctivitis, there remains solely, at the end of nine months, a fine papillary condition of the upper tarsal conjunctiva; one of these two cases has had two relapses or infections.

It may be mentioned here in passing, that of the seventy-five cases of inclusion conjunctivitis studied by us during the past five years, fifty-eight cases are still under observation of Dr. Cohen. Two of these still show a diffuse and linear cicatrization of practically the entire palpebral conjunctiva, but no corneal or other involvement is present. Two other cases of this group still show a fine papillary hypertrophy of both tarsal conjunctivæ, with no corneal or other complications. The remaining fifty-four cases have remained normal for the past four and a half years. Seventeen cases were observed for six months, and were normal when last seen.

Four patients with follicular conjunctivitis became infected with inclusions. The follicles previously present became enlarged and congested, and new papules and follicles appeared on the upper tarsal conjunctiva and the conjunctiva of the lower lid. These cases had the appearance of a severe type of inclusion conjunctivitis.

For several weeks after the beginning of the disease, the smears in all cases showed an abundance of epithelial cell inclusions. Toward the end of the disease, the number of inclusions gradually diminished. Yet after three months' duration, although no inclusions were demonstrable in the smears, the clinical aspect of the disease was usually still evident.

On October 23, 1914, one of the inclusion cases on the Island developed a slight conjunctival inflammation, which smears and cultures showed to be due to the Koch-Weeks bacillus. In the succeeding two weeks, as previously mentioned, additional cases of inclusion conjunctivitis contracted the Koch-Weeks bacillus infection. Conjunctival smears

and cultures examined weekly for the past several months showed at first the simultaneous presence of inclusion and Koch-Weeks bacilli. In the succeeding two months, the findings varied, at times only one of the two organisms being present; at other times, both. Possibly this was due to relapse or reinfection. In the next two months, although no organism was present in smears and cultures, there was still evidence of inflammation. Ultimately the conjunctiva became normal in all but one case. In this case there is still at the end of a year evidence of an inclusion conjunctivitis in the retrogressive stage.

In an adjoining ward, where mild follicular conjunctivitis cases were being treated, thirteen contracted the Koch-Weeks bacillus infection. Previous to the Koch-Weeks infection, all these cases showed over various areas of the conjunctiva follicles varying in number and size, and showing no inflammatory reaction. The first case in this ward developed on November 1, 1914. Only three of the sixteen cases escaped the Koch-Weeks infection. The Koch-Weeks bacillus was present in great numbers, but no other pathogenic organisms could be demonstrated throughout the entire course of the epidemic, which lasted six months.

The clinical manifestations in these follicular cases were as follows. There were slight œdema of the lids and conjunctival congestion with mucopurulent secretion. Both eyes became successively involved. The acute infection lasted six to eight weeks. At the end of this period, the original follicles were still present.

In one of the typical follicular cases it was difficult to determine whether the acute infection was due to the Koch-Weeks bacillus or to the epithelial inclusions. The symptoms were those of a severe inclusion conjunctivitis. But notwithstanding repeated examinations extending over two months, no inclusions could be found. Nevertheless, judging from the clinical course and from the fact that cell inclusions are often absent in the subacute stage, this and similar cases may be considered due to epithelial inclusions.

Two cases of interstitial keratitis in the Island Eye Ward and one at the Dispensary had normal conjunctivæ, when they suddenly developed an acute catarrhal conjunctivitis.

In the smears and cultures, which were taken weekly for four months, the Koch-Weeks bacillus was present, even at a time when the conjunctiva was free from inflammation. The inflammatory stage lasted from six to eight weeks. Inclusions could at no time be demonstrated in the smears.

The clinical appearance in these cases was moderate œdema of the lids, and conjunctival congestion with mucopurulent secretion. In one case there was present a small sclero-conjunctival hemorrhage with a small phlyctena. Conjunctival furrows or folds also appeared in those cases when the congestion diminished.

As regards the last group in the table, two cases at first showed the Koch-Weeks bacillus in the conjunctival smears. Later, inclusion cells made their appearance, associated with their clinical manifestations. In the first case there was present a mild catarrhal conjunctivitis, papules being present on the lower tarsal conjunctiva alone. In this respect it differed from other inclusion cases where the papules are present also on the upper tarsal conjunctiva. It is possible that the lesions at times can be detected only microscopically. A second examination in this case revealed the epithelial inclusions. Perhaps the inclusions were missed in making the first examination, as it is likely that in some stages of the disease only a few epithelial cells contain inclusions.

The second patient originally had a follicular conjunctivitis. He then contracted an acute catarrhal conjunctivitis, the secretion containing for three weeks the Koch-Weeks bacillus alone. At the end of this time, when the conjunctivitis improved, he again developed an acute conjunctival inflammation, and the smears showed numerous inclusions and a few Koch-Weeks bacilli. This case then followed the usual course of an inclusion conjunctivitis, the conjunctiva ultimately returning to a normal condition.

In a ward assigned to minor eye affections, where there were a few mild Koch-Weeks cases, a routine examination of the conjunctivæ of all the cases was made, in order to determine the presence of the Koch-Weeks bacillus. Two cases showed the Koch-Weeks bacilli, in spite of the fact that the conjunctivæ were entirely normal throughout the whole period. In another case in the same ward degenerated Koch-

Weeks bacilli were found in the conjunctival smears. This case then developed an acute inflammation, showing all the symptoms of an inclusion conjunctivitis with the added findings of epithelial cell inclusions in the smear examination.

In our routine examinations made during the past five years Koch-Weeks bacilli have rarely been found. Recently we encountered eight cases of pneumococcal conjunctivitis. Clinically these cases resembled acute catarrhal conjunctivitis, due to the Koch-Weeks bacillus, and were so diagnosed before the smears and cultures showed the presence of numerous pneumococci without any Koch-Weeks bacillus. The conjunctival secretions in these cases were typically serous, being also associated with diffuse congestion. The duration was from one to two weeks. One of the patients, included already in this paper, had originally an inclusion conjunctivitis. Later, he contracted a Koch-Weeks bacillus infection. After four weeks, when apparently cured, another acute inflammation appeared, which was due to the pneumococcus. This infection was followed by the appearance of inclusion conjunctivitis, the smears showing both pneumococcus and inclusion cells. At present, the clinical symptoms are those of an inclusion conjunctivitis in a state of relapse or reinfection.

The treatment adopted in all these cases was irrigation of the conjunctival sac with a saturated solution of boric acid every two hours, and the application to the conjunctiva, once a day, of a silver nitrate solution, until the acute symptoms had subsided, when irrigation alone was continued. This method of treatment had practically but little effect on the course of the inclusion cases, whereas in the Koch-Weeks infections decided improvement was observable. The disappearance of the organisms from the smears and culture was, however, not at all influenced by the application of the silver nitrate.

BACTERIOLOGICAL AND EXPERIMENTAL STUDIES OF THE PRESENT EPIDEMIC.[1]

Our technic for obtaining smears and cultures was as follows. The upper lid was everted and then the short end of

[1] This part of the work was carried out at the Rockefeller Institute.

a sterile slide was gently rubbed over the upper tarsal conjunctiva where there is least likelihood of contamination. By means of a platinum loop, a part of the scrapings removed was used for the purpose of cultivation and animal experiments. The remainder of the materials on the slide was transferred to another slide where a thin spread was made, and examined by means of the Giemsa or Gram stain.

Since our problem deals with the Koch-Weeks infection, the culture media employed were chiefly blood agar and tissue ascitic fluid. The cultural findings are summarized below.

In the present epidemic it was noticed that various strains of the so-called Koch-Weeks bacilli or hæmoglobinophilic organisms in cultures varied in size to such an extent that they may be divided into a thin (Figs. 4 to 7), a medium (Figs. 8 to 11, 20 to 23), and a coarse type (Figs. 16 to 19), according to their morphology. The coarse variety were bacillary or coccoid (Figs. 12 to 15) and resembled culturally and morphologically some strains of *Bacillus influenzæ* (Figs. 24 and 25) derived from the respiratory organs; while the thinner variety was much narrower and often shorter. After several days' cultivation at 37° C. on blood agar, some of the organisms became somewhat granular and unevenly stainable; when found in large masses they often assumed the appearance of the granular forms of the so-called trachoma bodies (Figs. 5 to 7, 9 to 11, 13 to 15, 17 to 19). But these granules did not break down so as to approach the minuteness of the elementary bodies (Figs. 32, 33, 36, 39, 43, 45) which are found in the uncomplicated cases of inclusion conjunctivitis or trachoma. The degenerated or involuted bacilli do not have the sharp contour of the elementary granules. Transplants made from these somewhat degenerated granular masses of the hæmoglobinophilic organisms give a good growth of typical bacillary or semicoccoid forms. The character of the colonies on the blood agar is also distinctive, in that they remain minute, sharply elevated with a pointed top, and appear more grayish than the colonies of the influenza type varieties, which show a tendency to spread and are of a more dewy aspect. It is quite possible that the minute type constitutes a group by itself, but further study is required to determine its relation to the other groups. At all events several strains of influenza

bacillus[1] derived from cases of meningitis and pneumonia appear quite different from the conjunctivitis strains in their morphological features (Figs. 24 to 28). The frequency with which organisms of different type were found in the present epidemic is shown in Table II.

TABLE II.

Organisms present.	Types.		
	Coarse.	Medium.	Thin.
1. Epithelial inclusions first, with subsequent Koch-Weeks infection (17 cases)	1	7	9
2. Koch-Weeks bacillus alone (13 cases)..	1	10	2
3. Koch-Weeks bacillus first, with subsequent appearance of inclusions(2 cases)	1 (round type)	1	

As will be seen from Table II., the thin variety occurred very frequently in the cases in which the patients had been previously infected with the inclusions. In the simple Koch-Weeks cases the medium was predominating. It is significant that in the smears taken from the cases infected with the inclusions and Koch-Weeks bacilli it is not difficult to differentiate the initial bodies of the former from the bacilli, because the initial bodies are much larger and oval in shape, and take up a deeper blue stain. Fig. 3 shows a typical inclusion near the nucleus and some Koch-Weeks bacilli in the same cell. The hæmoglobinophilic bacilli are seen to be taken up by the polynuclear leucocytes more frequently than by the epithelial cells (Fig. 1), while the inclusion bodies are chiefly epithelial and seldom leucocytic in nature (Figs. 29 to 45). So far as could be ascertained in stained specimens the bacilli remain well preserved within the cells and do not seem to disintegrate into granules (Fig. 2). On the other hand, the initial bodies are often ill defined and do not show any distinct bacillary forms. They are far more pleomorphic than the hæmoglobinophilic bacilli (Figs. 29, 34, 37, 40, 41, 43, 46, and 47).

[1] We are indebted to Dr. Martha Wollstein for these strains, for which we here wish to express our thanks.

The isolation of the hæmoglobinophilic organisms, irrespective of the type, was easily accomplished by means of the blood agar; but in many instances the preliminary microscopical search in the smears failed to demonstrate the presence of a few organisms, until they could be found by the cultural procedure. It must be understood that in cases where the inclusions only were found, repeated efforts to isolate the hæmoglobinophilic organisms were made, with, however, invariably negative results. In this connection it may be added that a number of cases clinically diagnosed as trachoma was also studied in order to see if any hæmoglobinophilic organism could be isolated. The results were uniformly negative. A few cases were also found in the same wards, which may have been infected through contact with the inclusion cases and which, although carefully observed from the earliest stage of the disease, did not at any period show the presence of any hæmoglobinophilic bacilli.

Transmission of the Inclusion Bodies to Animals.—In order to find out whether the inclusion virus ever exists in the form of a hæmoglobinophilic bacillus, scrapings from the conjunctivæ of these cases were inoculated into the conjunctiva of a baboon. Prior to the inoculation the conjunctiva of the baboon was examined to ensure the absence of such a bacillus. Within seventy-two hours after the inoculation the conjunctiva showed moderate congestion, œdema, and a few minute papules; a small quantity of a mucopurulent discharge was present at the inner canthus. The smears and culture were made from the conjunctival scrapings. The result showed that there were numerous cell inclusions, but no hæmoglobinophilic organisms[1] (Figs. 46 to 51). The examinations were continued regularly for a period of two weeks and the results were unvarying. As was previously shown by various investigators, the von Prowazek bodies are transmissible to higher apes, but the hæmoglobinophilic or Koch-Weeks bacillus is not.

Attempts to Produce Koch-Weeks Bacillus Conjunctivitis in Animals.—Several attempts were made to transfer the hæmoglobinophilic organisms to the conjunctivæ of rabbits

[1] Cultures made after twenty-four and forty-eight hours were also negative in regard to the hæmoglobinophilic bacillus.

(young and adult) and monkeys (baboon and several *Macacus rhesus*), by introducing several loopfuls of the twenty-four-hour blood agar cultures of different strains into moderately abraded surfaces of conjunctivæ. The results were completely negative, except in the case of one rabbit, where the organism was still recoverable after twenty-four hours. It seems remarkable that such a large quantity of pure cultures of freshly isolated strains of these organisms from the cases where the inclusions were also present should fail to reproduce the conjunctivitis, in view of the fact that a comparatively small number of the inclusions as contained in the scrapings from a patient can readily reproduce the inclusion conjunctivitis.

From this a conclusion may be warranted that in a conjunctivitis where the inclusion and the hæmoglobinophilic bacilli are simultaneously present, two pathogenic factors can be separated by means of transmission of the material into the conjunctiva of a suitable animal (baboon) in which the inclusion virus alone implants itself upon the new host, while the bacilli quickly disappear from the inoculated conjunctiva. For man both organisms are pathogenic, but for the baboon only the inclusion virus is capable of producing infection.

On the other hand, this may not exclude the possibility, as asserted by Williams and her associates, that a conjunctivitis due to the Koch-Weeks bacillus may also show some cell inclusions; since under certain experimental conditions a very suggestive phenomenon, to be related below, has been observed.

Attempts to Produce the Koch-Weeks Bacillus Epithelial Inclusions in Animals.—In order to determine experimentally whether or not the hæmoglobinophilic bacilli when taken up by epithelial cells will undergo the morphological changes which lead to the formation of so-called inclusion bodies, an intratesticular inoculation of the rabbit with pure cultures of the hæmoglobinophilic organisms isolated from the cases already mentioned was resorted to. The local reaction which follows consists of œdema of the scrotum and induration of the testicular parenchyma within twenty-four hours. The œdema gradually disappears within the next few days, while

the testicular induration remains more or less the same for about five days, after which it commences to recede. In some instances the rabbit succumbed to septicæmia and probably to intoxication as a result of the introduction of the cultures. The organs were removed at intervals of twenty-four hours, three days, and six days, and then fixed in sublimate alcohol, and stained by Giemsa's acetone method,[1] and, if overstained, treated with a ten per cent. solution of *Glycerinäthermischung* (Grübler) for a few minutes, as advocated by one of us.[2] The results show that the injection of the bacilli is followed by an intense leucocytosis, in which the polynuclears invade the tubules in groups. In the twenty-four-hour specimens the organisms are still well distributed along the interstitial spaces; in the three-day specimens one notices numerous masses of agglutinated bacilli here and there within the tubular lumina or along the connective tissue. These masses take on a purplish hue and appear granular in structure and indefinite in outline. They are on the point of disintegration. The granules within and about these bacterial masses are not so minute as to be mistaken for the elementary bodies of the inclusion. In the six-day specimens some clumped bacteria were found within the polynuclear leucocytes, but a diligent search failed to show any typical epithelial cell inclusions. In these six-day preparations the number of the bacterial clumps is smaller than at an earlier period. There were no granules small enough to be regarded as the typical elementary granules. While a careful comparison of the inclusions and the bacterial clumps just referred to will reveal the difference between them, this is not always easy to accomplish (Figs. 53 to 55). Fig. 52 shows a mass of granules from a case of mixed infection of Koch-Weeks bacilli and the von Prowazek inclusions, and it appears difficult to determine whether they represent the degenerated bacilli of the inclusion granules. The deep stain of the mass and the absence of any free bacilli around it seem to indicate that it belongs to the latter kind. It is also possible that some of the clumped granules found in the conjunctival smears from cases of

[1] Giemsa, G., *Deutsch. med. Wchnschr.*, 1909, xxxv., 1752.
[2] Noguchi and Cohen, *Proc. N. Y. Path. Soc.*, 1910, x., 20.

Koch-Weeks infection might have been interpreted as the cell inclusions and classified with the von Prowazek inclusions.

Attempts to Transmit the Inclusion Bodies to a Parenchymatous Organ in Animals.—Efforts were also made to transmit the von Prowazek bodies from uncomplicated inclusion cases to the testicles of rabbits, since this organ offers an excellent medium of growth to various highly parasitic organisms which otherwise cannot be easily cultivated.[1] The scrapings of conjunctivæ from four different patients were inoculated into the testicles of eight rabbits; but in spite of the large number of the inclusions contained in the conjunctival scrapings used for this purpose, no success was obtained along this line. The testicles showed within twenty-four hours some induration and œdema, but after a few days resumed their normal condition. Tissues removed after twenty-four hours, three days, and six days failed to show any cell inclusions when examined in smears and sections. No hæmoglobinophilic organism was found in cultures made from the tissues. This negative finding also tends to strengthen the view that the cell inclusions found in these cases were not the Koch-Weeks bacilli, for if they had been it would have resulted in the production of Koch-Weeks orchitis.

CONCLUSIONS.

1. There are cases in which epithelial cell inclusions may alone be present in the conjunctival smears. In such cases no other pathogenic organisms, such as the Koch-Weeks bacillus or the pneumococcus, can be demonstrated in smears or cultures.

2. The conjunctiva can become simultaneously infected with the inclusion bodies and Koch-Weeks bacilli or other organisms.

3. In cases of acute or subacute conjunctival inflammations due to mixed infections the clinical features of each infection may be present. The course of the inflammation is, however, more prolonged.

4. Within recent years, the Koch-Weeks bacillus has only seldom been found in our routine examinations.

[1] Noguchi, H., *Jour. Exper. Med.*, 1915, xxi., 539.

5. The epidemic studied was of a severe type.

6. Clinically it is practically impossible to distinguish pneumococcal conjunctivitis from the Koch-Weeks conjunctivitis. Bacteriological examination of smears and cultures is the only means by which the etiological diagnosis can be definitely established.

7. Conjunctivæ of certain species of monkeys are susceptible to the von Prowazek inclusion bodies, but not to the hæmoglobinophilic bacilli isolated from cases of epidemic conjunctivitis.

8. The injection of conjunctival scrapings containing the von Prowazek cell inclusions into the testicles of rabbits produces no cell inclusions in the latter, while the injection of a pure culture of the hæmoglobinophilic bacilli causes an acute inflammation accompanied by numerous clumps of the organisms, simulating the von Prowazek bodies at certain stages of their evolution.

9. There exists an apparent morphological similarity between the degenerated forms of this variety of the hæmoglobinophilic bacilli and the cell inclusions, both in cultures and in experimental orchitis in the rabbit. But, as a rule, the elementary bodies of the latter are much smaller and more sharply defined than the smallest granules of the former, while the initial bodies are bigger, more intensely stainable, and less definite in their contour than the hæmoglobinophilic bacilli found in the infected conjunctivæ.

EXPLANATION OF TEXT-PLATES.

All the photographs were made from film preparations, except where otherwise stated. They were stained with Giemsa. The enlargement is uniformly 1000 diameters.

TEXT-PLATE IV.

FIG. 1.—This shows a mass of the Koch-Weeks bacilli in a film preparation from an uncomplicated case of Koch-Weeks conjunctivitis. The bacilli belong to the thin type and some scattered examples appear coccoid. By focusing, the mass is seen to be composed of numerous well defined bacilli which cannot be confused with the inclusion granules.

FIG. 2.—An epithelial cell from a case of uncomplicated Koch-Weeks conjunctivitis. Around the periphery of the cell and near the nucleus along the lower border numerous bacilli are seen to be attached to, or contained

within, the cell body. There is, however, no difficulty in recognizing the bacilli as such in this instance.

FIG. 3.—A film preparation from a case of mixed infection of the Koch-Weeks and the inclusion organisms. In the field the epithelial cell is seen to contain a densely stained mass of the initial bodies near the upper right border of the nucleus and numerous Koch-Weeks bacilli to the left, especially where there is a leucocyte.

FIG. 4.—This represents a twenty-four-hour-old pure culture of a thin type of the Koch-Weeks bacilli on blood agar.

FIGS. 5, 6, and 7.—The appearance of the same organism as in Fig. 4 after three days, five days, and eight days, respectively, on the same medium at 37° C.

FIGS. 8, 9, 10, and 11.—These represent the appearance of a medium type strain of the Koch-Weeks bacilli after twenty-four hours, three days, five days, and eight days, respectively, on blood agar at 37° C.

FIGS. 12, 13, 14, and 15.—These show the appearance of a strain of round or coccoid type of the Koch-Weeks bacilli in a twenty-four-hour, three-day, five-day, and eight-day growth on blood agar, respectively.

FIGS. 16, 17, 18, and 19.—These show the appearance of a coarse strain of the Koch-Weeks bacilli in a twenty-four-hour, three-day, five-day, and eight-day growth on blood agar, respectively. This strain resembles *B. influenzæ* more than the others.

FIGS. 20, 21, 22, and 23.—These show four different strains of the Koch-Weeks bacilli isolated from the mixed infection cases in the present epidemic. They belong to the medium type.

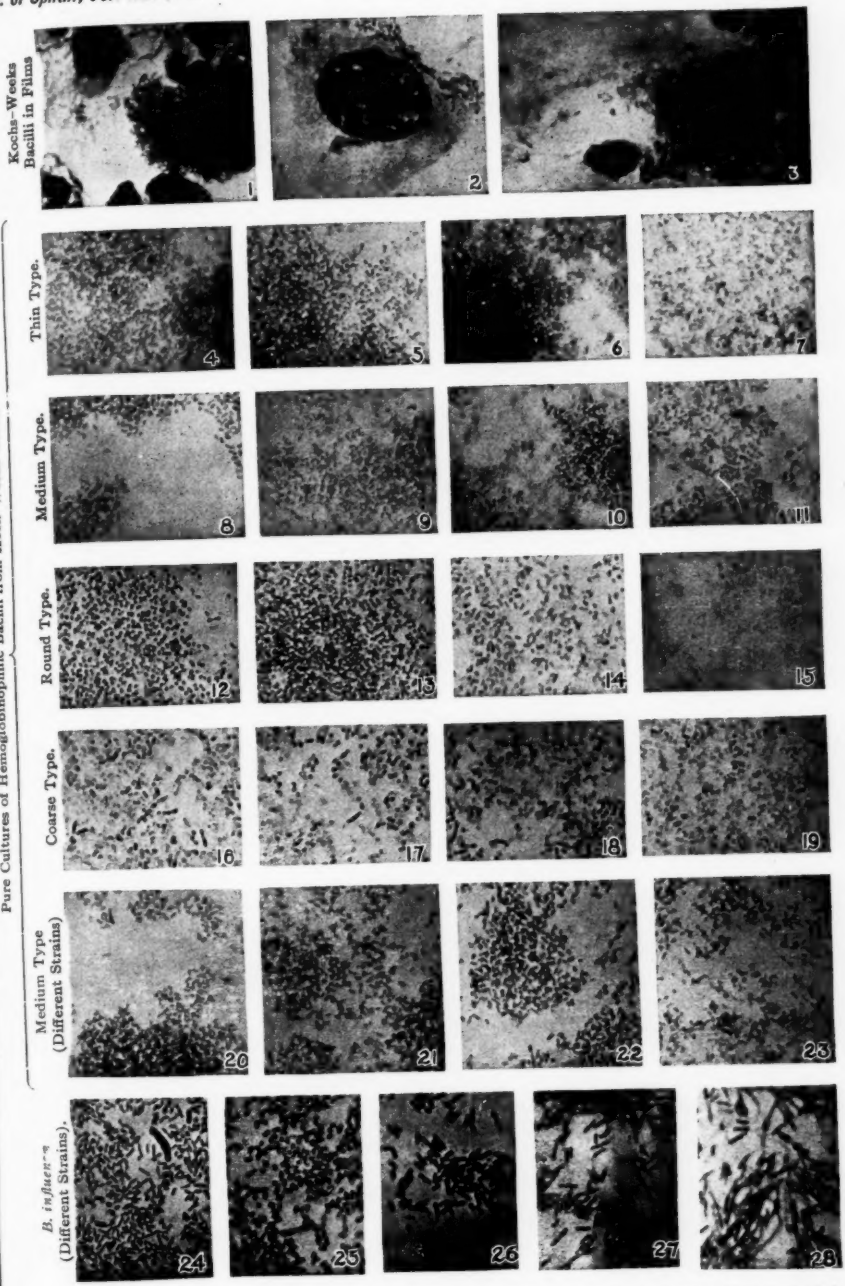
FIGS. 24, 25, and 26.—These show twenty-four-hour growths of three different strains of *B. influenzæ* isolated from cases of pneumonia.

FIGS. 27 and 28. These show twenty-four-hour growths of two different strains of *B. influenzæ* isolated from cases of meningitis. Both strains were kept on artificial media for several years and tend to form threads more than they originally did (Wolstein).

TEXT-PLATE V.

FIGS. 29, 30, 31, 32, and 33.—These show the inclusion at various stages of evolution in the film preparations made from a case (Tho.) of inclusion conjunctivitis. This patient had been suffering from the inclusion conjunctivitis when he was superinfected with the Koch-Weeks bacilli, which, in due course, disappeared from his conjunctiva leaving the original condition little affected. These inclusions shown here were found in the conjunctiva long after the Koch-Weeks bacilli had disappeared in smears or in cultures. The minute elementary bodies in Figs. 32 and 33 bear no resemblance to the degenerated forms of the Koch-Weeks bacilli.

FIGS. 34, 35, and 36.—These represent the inclusion bodies in an uncomplicated case (Sh.), that is, without any association with the Koch-Weeks bacilli. The character of the initial bodies in Fig. 34 would definitely dispose of any suggestion as to their being the Koch-Weeks bacilli. Fig. 36 shows a still unburst intracellular aggregation of the elementary bodies.



(Noguchi and Cohen; Epidemic of Koch-Weeks Bacillus Conjunctivitis.)

Inclusion Bodies in Film Preparations from Uncomplicated Cases.

Arch. o

Inclusion Bodies in Film Preparations from Uncomplicated Cases.

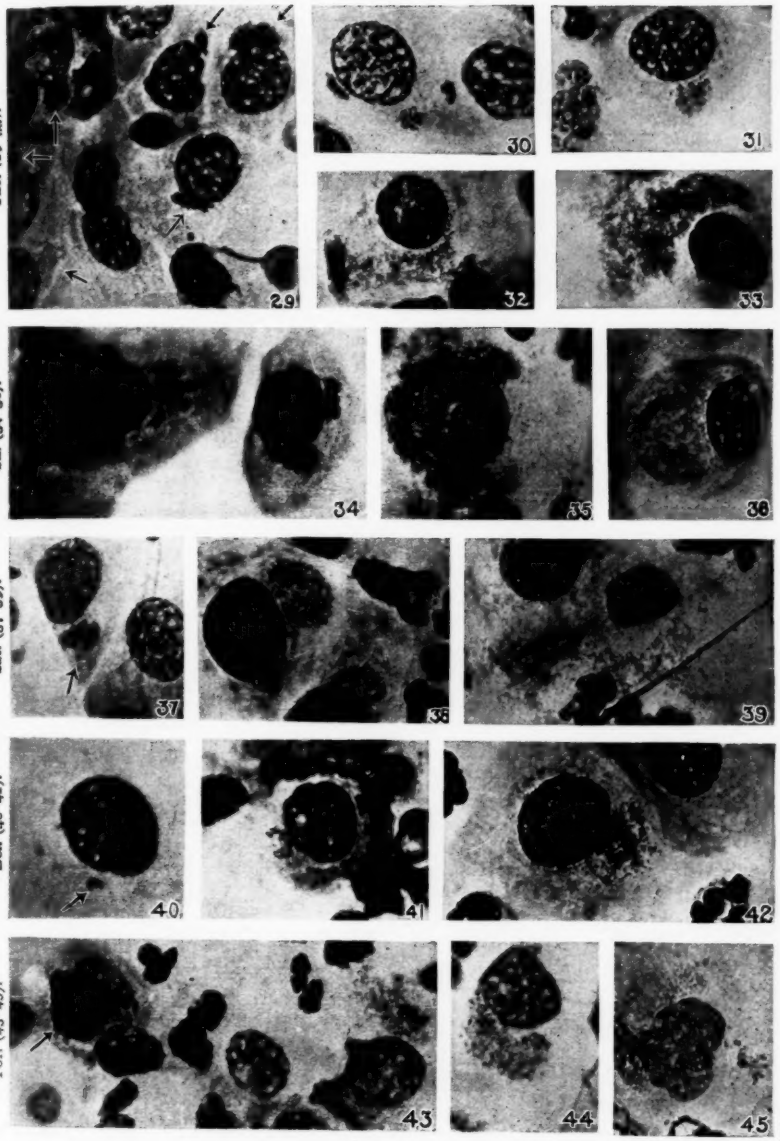
Tho. (29-33).

Sb. (34-36).

Sat. (37-39).

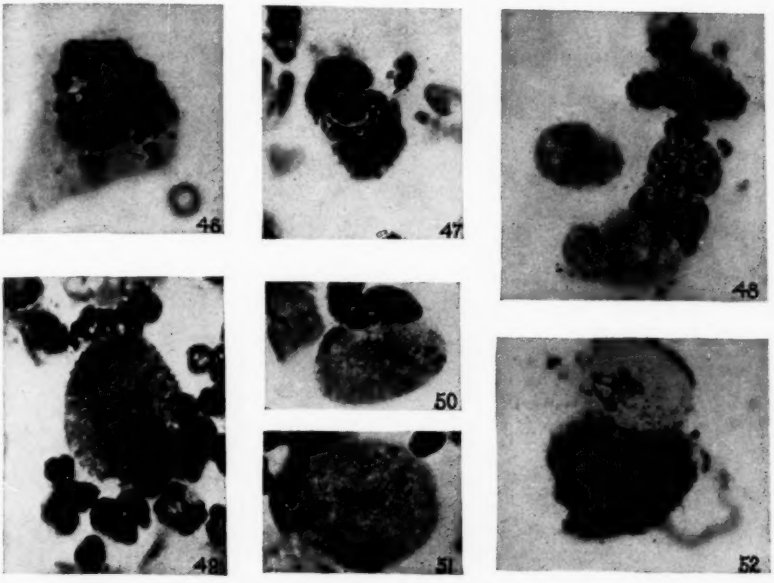
Bol. (40-42).

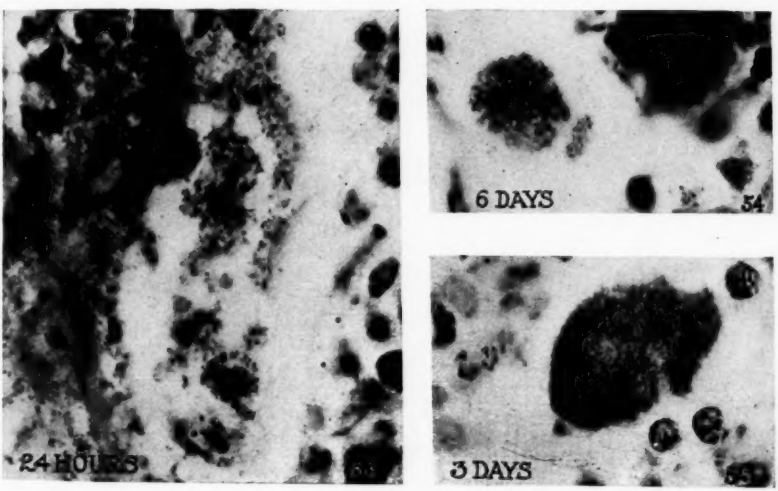
For. (43-45).



(Noguchi and Cohen: Epidemic of Koch-Weeks Bacillus Conjunctivitis.)

Experimental Inclusion Conjunctivitis in Baboon (46–51).





Experimental Koch–Weeks Orchitis in Rabbit (53–55).

(Noguchi and Cohen: Epidemic of Koch–Weeks Bacillus Conjunctivitis.)

FIGS. 37, 38, and 39.—Inclusions from another case (Sat.) of pure inclusion conjunctivitis.

FIGS. 40, 41, and 42.—Inclusions from a case (Bol.) of trachoma. The initial bodies as shown in Fig. 40 are exceptionally coarse.

FIGS. 43, 44, and 45.—Inclusions from an uncomplicated case (For.) of inclusion conjunctivitis. In Fig. 43 a thick mass of the initial bodies is seen to embrace the left side of the nucleus of a cell on the left, while on the right numerous elementary granules are scattered around the cell below. Figs. 44 and 45 show the steps of evolution of the inclusion bodies.

TEXT-PLATE VI.

FIGS. 46, 47, 48, 49, 50, and 51.—Different stages of the evolution of the inclusion bodies and the conjunctiva of a baboon experimentally infected with the scrapings of the affected conjunctivæ of man. The structure of the initial bodies in Figs. 46, 47, and 48 is quite peculiar and reveals no definite bacillary outlines. They are deeply stained, almost amorphous, and show indistinct coarse granules within the mass. The elementary bodies are even more minute than those in the human cases, as will be seen in the cell occupying the lower half of Fig. 48. In the same figure one sees an irregularly shaped mass of the initial bodies at the upper corner. In Figs. 49, 50, and 51 are shown the enormously distended cells in which the inclusion granules are rapidly multiplying.

FIG. 52.—This shows a dense mass of deeply stained granules in a film preparation from a case of mixed infection. This mass appears to be more of the von Prowazek type of body than of the degenerated Koch-Weeks bacilli.

FIGS. 53, 54, and 55.—Sections of the rabbits' testicles inoculated with pure cultures of Koch-Weeks bacilli isolated from the mixed infection cases during the present epidemic. Fig. 53 shows that the organisms assume a coccobacillary form when examined within twenty-four hours after they were injected into the testicle. In photography they appear somewhat like the initial bodies in the inclusion cases, yet in actual examination of the preparations they are not difficult to distinguish as the Koch-Weeks bacilli. They are freely capable of cultivation when a minute portion of the testicle is transferred to the blood agar, while this is not the case with the initial bodies of the inclusion organism. In Figs. 54 and 55 the sections removed after three and six days, respectively, are shown in which large masses of agglutinated and somewhat ill-defined bacilli are found in clear spaces within the lumen of the testicular tubules. They, too, present an appearance not unlike that of the inclusion organism found in experimental conjunctivitis in monkeys, as well as in an epidemic in man. Here, again, a culture on blood agar is quickly differentiated from the other kind, as the Koch-Weeks bacilli at this stage still thrive very well in culture, while the pure inclusion material does not give any growth with the hæmoglobinophilic bacilli.

CLINICAL PATHOLOGY OF OCULAR MUSCLE PARALYSIS.

A CRITIQUE OF THE MORE RECENT LITERATURE ON OCULAR MOVEMENTS, WITH SPECIAL REFERENCE TO THE STUDY OF THE CONTROL OF OCULAR MUSCLES.

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A CRITICAL analysis of the more recent literature on ocular movements impresses one with the development of the science, and shows indications of upward trend which are worthy of note.[1]

ANATOMY AND PHYSIOLOGY

The fundamentals—anatomy and physiology—as related to ocular movements are peculiarly associated with pathology, since it is largely by the pathologic lesions—either experimental or spontaneous—that the functions are studied.

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[1] This critique has not been undertaken with a sense of any special ability to pass on the work of others, either because of qualifications or by reason of experience. It is submitted with a consciousness of its incompleteness and of its shortcomings. A critique offers manifestly some embarrassment to its author, since such an analysis shows inevitably occasional error of statement or of interpretation, or in method of study or report, which must needs be noted if the maximum good is to be accomplished. It is possible to review in this paper only that portion of the literature of ocular movements which bears on the clinical pathology and the application thereof to the ever present and unsolved problem of localization of control. The interpretation of these findings as to their bearing on the problem of primary causation of nonparalytic muscular imbalance will prove an interesting subject to work on, and will form the basis of another chapter on ocular balance and motility.

From a broad point of view it may be seen that the volitional nature of the frontal, the reflex nature of the hind and mid-brain, and the specific or individual nature of the lower levels of nuclear control are becoming more evident with recent findings.

The nervous system naturally falls, therefore, into the three classical levels of functional activity, and it is to these levels we wish to direct our study.

As a preliminary to the consideration, it might be well to define just what is comprehended by these levels of control according to our plan.

The *first* or *lower level* comprises the nuclei and nerves of the ocular muscles, the specific muscles concerned in ocular balance and motility.

This neuromuscular mechanism is phylogenetically fundamental, and the variation in the relations of nuclei to afferent fibers is consistent with a fixed plan, the crossing or partial crossing being governed by the developments of binocular vision.

Kidd (1) has added to this subject by his study. He states that there is anatomic, experimental, and pathologic proof of the existence of direct (uncrossed) trochlear fibers from the lower forms up to man, in whom the direct fibers are relatively few, since the crossed have increased enormously in the phylogenetic development.

He also claims similar proof of the existence of crossed oculomotor roots and fibers. The direct and crossed fibers of the oculomotor nerve are distributed as follows: The inferior oblique receives a few direct and most of the crossed fibers; the superior and inferior recti, mainly the direct, and the internal rectus exclusively crossed fibers, all of which come from the contracentral oculomotor nucleus. The external rectus receives wholly direct fibers. There are no fibers in man going from the abducens nucleus via the posterior longitudinal bundle to the contracentral nucleus or root.

Also a lesion strictly limited to the abducens nucleus gives the same signs as the nerve root, namely, a palsy of the external rectus, whereas a complete cellular lesion of the trochlear nuclei gives bilateral signs, namely, slight paresis of the homo-

lateral superior oblique with almost complete paralysis of the contralateral superior oblique.

The nuclei for the ocular muscles are present in lower forms and persist throughout, and are oriented in man, as you well know, beneath the floor of the anterior portion of the fourth ventricle for the sixth nerve, in the floor of the iter, level with the anterior half of the hind quadrigeminal bodies, for the fourth nerve nucleus, and for the third nerve also in the floor of the iter just above the fourth and corresponding in level with the groove between the anterior and posterior quadrigemina. This nucleus, which is about 1*cm* long, extends beyond the iter into the hind portion of the third ventricle.

The *second* or *midlevel* is that of reflex control—also fundamental phylogenetically, but varying as to the site of control centers between higher and lower forms.

The quadrigemina bodies, as shown by many observers, have complicated functions in all the lower animal forms, but become of less relative importance as binocular use of the eyes develops. Hence, as the cortex becomes developed coincident with the demands for increasing binocular vision, the importance of these bodies is diminished.

Fisher (2) thinks and offers proof that lesions of the quadrigemina may sometimes exist in man without affections of vision, and that these bodies must not be regarded as containing subsidiary coördinating centers. And though ocular paralyses are among the most conspicuous of the symptoms in cases of lesions of the quadrigemina, such disorders of eye movement are considered to be distant symptoms.

The geniculates take up some of the functions of the quadrigemina in higher forms and doubtless in man constitute, with the quadrigemina and the pulvinar, substations for the oculomotor activity. But, inasmuch as phylogenetic study shows their slow diminution in relative importance, we must look still higher for the more highly developed reflex coördinated control in man.

Sherrington proved that stimulation of the occipital cortex produced movements of the eyes, not by utilizing association fibers passing to the Rolandic area, but by direct stimulation of the nuclei of the lower level and through some of the corticifugal fibers; and these are associated with the afferent visual

fibers passing to the same region of the cortex and lying in the more posterior portion of the hind limb of the internal capsule, whereas fibers passing to the base from the Rolandic area of the cortex occupy the most forward position in the anterior limb of the internal capsule.

It must not be forgotten that as long ago as 1888, Shafer (3) showed that the dorsal portion of the visual areas in the occipital lobes is associated with the upper portion of the retina, the eyes being turned downward when this area was stimulated, and this was interpreted as a movement of the eyes which would bring the upper retina into the center of the field of vision—a highly developed reflex coördination. When the stimulus was applied to the ventral portion of the area, corresponding upward movements of the eyes occurred; and the corresponding relation holds between the outer and inner retinal segments and the lateral and mesial portions of the cortical area in the occipital lobe. These movements occur in both eyes, even when but one side is stimulated, which shows that the two retinal fields are superposed in the visual cortex of the occipital lobes of each hemisphere, as stated by Howell (4).

The excessive number of axons in the association between the lower level centers—the nuclei—and the occipital lobes through the optic radiations, has also forced the conclusion that the occipital cortex has other functions than carrying visual impulses.

Burkholder (5) believes this is the connection for the faculty of fusion located in the occipital lobes. As will be later further shown, the association between the occipital cortex and the nuclei is independent of the third or high level of control.

The second level of control would therefore be found in the quadrigemina, the geniculates, the pulvinar, and in the occipital cortex.

The *third* or *high level* is that of volition or voluntary control. The ocular movement centers of the forebrain are known to lie in quite well defined areas, namely, immediately anterior to the precentral sulcus— that is, in the ascending frontal convolution and below the centers for the upper extremities.

On destruction of these areas, by removal of the lobes, as

done by Ferrier and Aldren Turner (6), there is conjugate deviation toward the lesion, but without ptosis or lid involvement. Irritation of these areas in monkeys, on the other hand, produced conjugate deviation to the opposite side.

Shafer (3) showed also very precise effects of ocular muscle stimulation through areas on both the temporal and occipital cortex—conjugate actions. He also showed, which is of importance, by dissociating the occipital and frontal areas by severing all the association fibers, that the connections of both these cortical areas with the cell groups controlling the muscles are independent. This demonstration has been overlooked by authors dealing with this subject. So we may conclude that voluntary impulses (volitional control) and reflex stimulation through vision are probably independent. Recovery occurring after the initial conjugate paralysis which follows removal of the lobes shows that while possibly dominant, the high level is not fundamental, since the reflex or second level itself becomes dominant and the balance is restored after a time.

Of most significance is Roux's (7) citation of several cases from literature, in which movements could not be produced by voluntary impulses to the tongue, lip, and face and eye muscles, but were produced promptly by reflex impressions from the retina. Such a condition, Roux says, is caused when both anterior frontal centers are incapable of function. See also the study of Durand and Jacquet (8).

Referring again to the work of Shafer (3) which went to prove what still seems probable, that the control fibers for the nuclei of the third, fourth, and sixth cranial nerves come from and are connected by neurons with cell groups in the cortex of both the occipital and forebrain, it was found that several hundredths of a second less were required for reaction from stimulation in the frontal region than when applied elsewhere. The voluntary control is then doubtless more direct than the reflex, and thus dominant.[1]

Again, Barnes's (9) conclusion is that the action of stimulus to the cortex is always conjugate; that stimulation of the middle level probably also induces conjugate action, while

[1] It would seem reasonable to speak of the second or midlevel as uncontrolled with reference to reflexes from the retina and voluntary to stimulation from the third or high level.

stimulation of the lowest level or nuclei results in independent ocular movements.

Of interest also are the works of Cestan (10), Hōgyes (11), Stumpf (12), Bartels (13), and Marina (14).

Considering the general subject of centers and connecting tracts, and the levels of action and control, recent literature has fixed more definitely the localization of lesions with reference to the levels of nerve action. These conclusions are about as follows:

In the high and middle levels—Campbell (15)—disorders of conjugate action are due to lesions in the cortex, the corticifugal tract, the supranuclear centers or their afferent fibers.

Fixation is possible in some parts of the field.

These cases cover paralysis of convergence and divergence, and paralyses of the lateral and vertical—all types of conjugate action.

Roemer (16) says:

"We notice in the examination of such a case (paralysis of lateral conjugate movement) that the internus that is affected functionates very well in convergence, although it cannot act in the conjugate movement of abduction (lateral conjugate version). This is because the tract for the innervation of convergence separates from that running to the corpora quadrigemina which transmits the innervation for the lateral movements. This tract on the other hand may be affected alone, and then we have a paresis of convergence, while the lateral movement of the internus is intact. Such conjugate pareses, with or without conjugate deviation, occur with lesions in the crura cerebelli ad pontem, in the pons, in the corpora quadrigemina, and in the thalamus opticus."

Savage (17) put it in another way,[1] namely:

"Injury or disease of the cortex, involving any one of the nine —according to Maddox, twelve (*vide infra*)—conjugate centers will result in paralysis or paresis of one (?) muscle connected with each eye, but the paralysis will show itself in the absence of verting power, with no loss of duction power.

"If the third conjugate center alone is involved, there can be

[1] Savage is right in speaking of verting power of the eye in contradistinction to ducting power.

no convergences, but because of freedom from disease of the fourth and fifth conjugate centers the two eyes can be made to turn harmoniously to the right or to the left.

"This high or midlevel paralysis has been termed 'paralysis of motion rather than of muscle.'"

Maddox (18) has added divergence control to his scheme of binocular innervations for creating the binocular eye, and his table of twelve binocular innervations is of interest here, although it concerns more the consideration of the phorias. This table should be borne in mind also with reference to Maddox's case of seesaw nystagmus, which will be detailed later.

MADDOX'S TABLE OF BINOCULAR INNERVATIONS.

KINGDOM I, FOR CREATING THE BINOCULAR EYE

Contrary	Horizontal	1.	Convergence
		2.	Divergence
	Vertical	3.	R Relative elevation over L.
		4.	L Relative elevation over R.
	Torsional	5.	Binocular intorsion
		6.	Binocular extorsion

KINGDOM II, FOR DIRECTING THE BINOCULAR EYE

Parallel	Horizontal	7.	Binocular dextroduction
		8.	Binocular levoduction
	Vertical	9.	Binocular elevation
		10.	Binocular depression
	Torsional	11.	Binocular dextrotorsion
		12.	Binocular levotorsion

Zentmayer's (19) case is of special interest, and, with Duane's (20) four cases, furnishes convincing evidence of the existence of a distinct center for divergence—proof of Maddox's subdivision 2 of contrary horizontal movements control.

Terrien and Hillion (21), Pasetti (22), and Farnariere (23) have cases of convergence paralysis which, taken with those heretofore reported, add proof of the entity of a specific convergence-coördinating center—Maddox's first subdivision of contrary horizontal movements control.

Sachs (24) has recorded a case of paralysis of conjugate lateral eye movements, adding confirmation of Maddox's subdivisions 7 and 8 of parallel horizontal movements control.

Pelisser's (25) case of paralysis of vertical movements is wrongly titled, since it is purely a basal case.

Freund's (26) is a presentation of the subject of paralysis of vertical movements (proof of Maddox's divisions 9 and 10 of parallel vertical movement control) and is an extremely interesting one, because it offers also much needed proof of high level localization as well. His conclusions are as follows:

"Such an important function as the coördinated control of the eye must be represented in various places in the cortex. Physiologic research has, in fact, shown three centers in the cortex for associated eye movements:

"First: The function of the anterior (frontal) centers on each side, at the foot of the second frontal convolution, Tschermah and Roux agree, is to control *voluntary* head and eye movements, but they *do not respond to optical, acoustic, or tactile impressions.*

"Second: The posterior occipital center is formed by the mother cells of the visual center calcarine fissure and the area immediately around it and responds only to visual impressions which attract and hold attention and vision by reflex paths: therefore it is called the visual reflex tract.

"Third: The visual center in the temporal lobe reacts to auditory impressions.

"The result of physiologic research and, especially, the demonstration of a center for voluntary head and eye movements at the end of the second frontal convolution, is particularly important, for it shows a cortical disturbance at the foot of the ventral half of the second and the dorsal part of the third right frontal convolution in the cited case. It is easy to see that, by the destruction of this center, the right posterior and longitudinal bundle is deprived of a considerable part of its impulses from the cortex, and that this explains the failure of function of the right longitudinal bundle and, therefore, the permanence of the paralysis.

"In conclusion, I think we are justified in assuming that the lateral third of the posterior longitudinal bundle is concerned in the function of conjugate eye movements in the vertical

direction and suspect that Darkschewitsch's center (27) is the controlling place on which cortical impulses for upward and downward movement of the eye act.

"What has been said shows how many points have hitherto been neglected in the clinical observation of individual cases of ocular paralysis, and how necessary it is, in all such cases, to make an histological examination of all sections of the central nervous system."

See also Farnariere (23).

LESIONS OF THE LOWER LEVEL

Lesions of the nuclei and their nerves produce actual paralyses of ocular muscles, one or more, so that in contradistinction to lesions in higher levels, fixation is impossible and the resulting diplopia involves the whole visual field. As such a paralysis indicates definite nuclear or nerve lesion, the difficulties of diagnosis as to the level involved are minimized.

Given an isolated paralysis of a muscle supplied by any branch of the third nerve, as the internus alone, or the superior, the inferior rectus or the inferior oblique, we know it must be from either the nerve or its nucleus; and as the nuclei are so closely related in their origin, the probability is against a nuclear lesion affecting only one branch.

The exact determination of localization of lesions of ocular paralysis is influenced of course by the syndromes and not alone by the terminal signs. In first or lower level paralyses, study of the whole syndrome is of particular importance.

Shoemaker (28) distinguishes the following as signs of paralysis of basal—low level—origin: first, a number of nerves being affected in succession; second, a neuralgia of the trigeminal; visual disturbances due to atrophy of the optic nerve or temporal hemiopia, and lastly the paralysis of the olfactory. Nuclear lesions seldom paralyze single muscles, and the nuclei for the sphincter of the iris and the ciliary muscles not corresponding to the third nerve nucleus, an affection of the third nerve without myosis is thought to be nuclear.

Paralysis of the whole oculomotor nerve must still be either nuclear or an afferent nerve, and hence still in the lower level, with a more even chance as between the nucleus and the nerve.

Similarly, paralysis limited to the superior oblique may quite definitely be fixed in the fourth nerve and not nuclear if both sides are involved; but if one side only, it must be in the nucleus (?), since the nerves are so closely approximated over the valve of Vieussens that both would be affected by a nerve lesion.

On the other hand, paralysis limited to the external rectus may be definitely fixed in the nerve and not in the nucleus. Generalized ocular muscle paralysis points to nuclear lesions, but paralysis of the externus of one side with hemiplegia of the opposite side would orient the lesion in the upper ventral pons, while a similar paralysis of the externus with a facial paralysis orients the lesion in the intercerebral course of these nerves or in both nuclei, since they are in close proximity.

Given a third nerve paralysis and a hemiplegia of the opposite side (Weber's syndrome) (29), this fixes the lesion in the crus. If, instead of hemiplegia of the opposite side, there is a tremor of the arm of the opposite side, it is still fixed at the crus, but limited to the inner part (Benedikt's syndrome) (30). The externus is currently thought to be most frequently involved in meningeal and basal pressure cases and traumatism because it has the longest exposed course. The distinction between paralysis and migrainous ophthalmoplegia is usually easy in that in the latter condition the third nerve alone tends to be affected; the symptom, further, is unilateral, occurs as a sequel to an attack of hemicrania, and is in abeyance during the intervals between the attacks.

Fuchs (31) distinguishes as between intracranial and extracranial or orbital lesions, as effecting paralysis. The intracranial are those strictly within the cranial cavity, and the orbital those which are within or forward of the fissure through which the nerves enter the orbit. He subdivides the intracranial into cortical lesions, or those affecting the high levels, fascicular or those between the nuclei and the exit from the brain surface, and basal or nerve trunk lesions between the brain surface and the orbital apex.

Orbital paralyses under Fuchs's classification may be from tumors, traumatisms, specific or inflammatory conditions; and as to the latter, a secondary involvement of the nerves through the accessory sinuses of the nose has particularly to be kept

before us. Posey (32) and Berard, and Sargon and Bessiere (33) are valuable additions to this literature.

SUMMARY OF LOW-LEVEL CASES.

Study of the most recent case reports on low-level lesions, as recorded in the year books of the last three years, may be thus totaled:

A. 45 Cases of third nerve branch affections, divided as follows:
   - (*a*) 37 cases of total third nerve involvement.
   - (*b*) 1 case of inferior rectus.
   - (*c*) 3 cases of internus.
   - (*d*) 1 case of superior rectus.
   - (*e*) 3 cases of inferior oblique.

B. 41 cases of abducens, and

C. 19 cases of superior oblique paralyses.

105

Analysis of these cases as to causation brings out the following:

84 of the 105 cases were due to traumatism.
5 cases were due to lues.
3 unknown, though probably lues.
3 cases secondary to sinus disease.
3 cases non-specific systemic causes.
2 cases due to injection of alcohol into the Gasserian ganglion for neuralgia, and one each to sclerosis, diphtheria, otitic abscesses, and one following on spinal anæsthesia.

Of the 84 cases due to traumatism, 33 were of the whole third nerve, and 3 were of its branches, 30 were of the abducens, and 18 of the superior oblique. These statistics thus tend to controvert the frequently handed down statement that the abducens is the most frequently involved in traumatism. Also it is surprising to note the large number of cases of superior oblique involvement, quite contrary to previous statistics. The palsies from Gasserian ganglion injection and that from spinal anæsthesia are also worthy of note.

No estimates are possible as to what proportion of these were orbital and which intracranial, though all are low-level lesions. Neither is it possible to get reliable data as to the proportion between the nuclear and nerve lesions.

Müller's (34) study of his 49 cases is of interest.

"The diagnosis of lesion of the base was made in 25 cases; orbital paralysis, 21; both base and orbit, 3. The muscles affected were the abducens, 22, with 6 cured; oculomotor, 28, with 19 cured; trochlearis, 18, with 10 cured, giving a prognosis of recovery in one half the cases."

See also Bergmeister's (35) report on traumatic types.

OPHTHALMOPLEGIA.

In the literature corresponding to the foregoing period, there are recorded 11 cases of ophthalmoplegia, 4 of which were bilateral, and 7 unilateral.

Roemer (36) understands ophthalmoplegia as "a syndrome dependent upon a disease of the posterior and middle brain, that is, of the medulla oblongata, pons, floor of the aqueduct of Sylvius, and the posterior part of the third ventricle." He also emphasizes the fact—which is contrary to the recent findings of Kidd (see above)—that a complete unilateral ophthalmoplegia cannot be of nuclear origin, since Roemer considers it certain that the fibers of the trochlearis undergo a total decussation, while the crossing of the third nerve is only partial.

ETIOLOGY.

With reference to general diseases, that is, diseases of the central nervous system, Barnes's (9) conclusions are of value:

"Of the various diseases of the central nervous system, tabes dorsalis and general paralysis may cause any ocular nerve palsy, partial or complete, and the muscle affected tends in many cases to become absolutely paralyzed. Gummatous meningitis produces ocular palsies which tend to clear up on mercury. In disseminated sclerosis, partial ocular palsies are common; nystagmus nearly always occurs at some stage and tends to persist. Nystagmus also occurs in Friedreich's

ataxia and in some cases of syringomyelia. In the last named, ocular palsies occur if the gliosis affects the aqueductus Sylvii. In progressive muscular atrophy, amytrophic lateral sclerosis, and bulbar paralysis the eye muscles are usually unaffected. In polioencephalomyelitis (infant paralysis) severe ocular palsies occasionally occur from nuclear inflammation, or nystagmus from cerebellar and tract involvement. Myasthenia gravis gives rise to a paralysis which tends in the early stages of the disease to be temporary only and to recover after rest; as the essential condition here is one of rapid exhaustion of the muscles themselves, diplopia is the rule in the paralytic period."

AS TO LUES.

It has generally been handed down that ocular muscle paralyses are about equally divided as between specific and nonspecific cases.[1]

The recently evolved tests for lues tend to diminish the cases of rheumatic and other systemic—"idiopathic"—types, in favor of the luetic. In fact, in view of the improved methods of diagnosis, there are many reasons for discarding records older than three years with reference to this factor in causation. Since Wassermann's reaction has been known, the cases of positive diagnosis of lues have steadily increased. It is only, however, in the last two or three years—in fact with some observers only very recently—that the appreciation has come that the Wassermann of the blood may be negative, and is rather apt to be negative, in central nerve involvement, and that even the Wassermann of the spinal fluid, or the gold test, or the more recent luetin test may be negative in such cases.

The extensive investigation by Nonne, tests of the syphilitic nervous diseases, and his epoch-making work on the cerebrospinal fluid have revolutionized this study. Recently Nonne (37) has recorded the study of twenty-three cases of pupillary disturbance in syphilitic individuals which he has followed in detail, and on which he has formed definite conclusions. In

[1] It is probable that no such ratio as is shown in the foregoing table exists as between traumatic and nontraumatic cases, and the literature of neurology and of syphilis would probably tell a different story of percentages.

one group of these cases the cerebrospinal fluid was normal from the beginning. In another group the fluid was at first positive, later normal. One case reported by Nonne and Wohlwell (38) is that of a woman with syphilitic aortitis, in whom the only nervous symptom was loss of her pupillary light reaction, but who gave positive blood Wassermann and a negative spinal fluid. This case came to necropsy, and is known to have been syphilitic. In another case the right pupil was smaller than the left, and slightly reacted to light and was accompanied by paralysis of accommodation in the right eye, and in this all four reactions were positive, the spinal fluid being at first positive and later negative. In still another of Nonne's cases the spinal fluid was positive throughout.

E. P. Corson-White and S. D. W. Ludlum (39) is another reference of importance on this subject.

Some of the conclusions of Paul Fildes and James McIntosh (40) are particularly interesting here:

1. A positive reaction in the cerebrospinal fluid indicates a syphilitic lesion of the central nervous system.

2. In active untreated cases of dementia paralytica, tabes dorsalis, and cerebrospinal syphilis the reaction is positive, both in the serum and cerebrospinal fluid. In cerebral syphilis without involvement of the spinal cord, the cerebrospinal fluid is usually negative, except that in the latter cases the strength of the reaction has no certain diagnostic value.

3. The reaction is apt to be negative in the serum or cerebrospinal fluid in cases of hemiplegia, nonprogressive tabes dorsalis, and old lesions of the nervous system, especially in cases of stationary congenital syphilis. It is also usually negative in the serum—particularly in very recent nerve lesions in the late secondary period, and in the cerebrospinal fluid, particularly in treated cerebrospinal cases.

4. The occurrence of a positive reaction in one of the test fluids when the other is negative is so common that a reaction found negative in one fluid only is insufficient to exclude syphilis.

5. Pure "parasyphilitic" lesions do not respond satisfactorily to treatment, as shown by the slight effect on the reaction in the cerebrospinal fluid. Cerebrospinal lesions react very readily to treatment. The celerity with which the

reaction in the cerebrospinal fluid responds to treatment is thus an index as to whether the case is a parasyphilitic or a syphilitic.

6. The positive reaction in the serum is rapidly made negative by treatment in acute syphilis; but in chronic syphilis this effect is produced very slowly. In the cerebrospinal fluid, on the other hand, the rate of reduction of the reaction does not depend on the duration of the infection. Thus, in gummatous lesions of the central nervous system, as also in acute secondary cerebrospinal syphilis, the effect of treatment on the reaction in the cerebrospinal fluid is rapid.

These recent developments in the serology of syphilis show us, as I have said, how little credence may be placed on a negative diagnosis of syphilis in all but the most recent literature, and even then, unless the definite statement is made as to what tests have been applied, we can not conclude that the case is nonspecific.

It is important also to point out some of the evil effects which have been observed by syphilographers ensuing on the use of salvarsan and analogous preparations, and the dangers of marked provocative reactions occurring in syphilitic disease of the central nervous system.

A few cases wherein paralysis has been precipitated by the use of salvarsan have found their way into our literature (41). Full bibliography of this may be found in the work of Benario, Ehrlich's assistant, on "Salvarsan Complications." The fact that this type of drug will cause neuritis of the cranial nerves was early described by Finger of Vienna, who reported some fifteen cases of this type. The theory is that the salvarsan destroys all the treponemes available in the general body, but not in the cerebrospinal canal, since as shown by Goldmann (42) the choroid plexus is an impermeable filter against extraneous substances; so that in cases in which there is infection within the canal there results not only no improvement, but for some reason, on extraneous injection of these arsenic preparations, an intensified growth of organisms and a neuritis, particularly at the constricted bony points of exit of cranial nerves. This has come to be described under the general term of "neuro recurrence," and it is important for us to bear it in mind in connection with this form of therapy. It seems likely

that subarachnoid injection of the drug will meet this difficulty and that improvement in results will be forthcoming.

As to fracture paralyses, one reason for the frequent paralysis of the externus in fracture cases is the liability of fracture involving the middle or posterior fossa across the apex of the petrous bone.[1]

Roemer (43) says:

"The oculomotorius is paralyzed in injuries of the skull much more rarely than the abducens and the facialis. In such a paralysis we must think primarily of a basal injury. . . . When the oculomotorius and abducens are simultaneously paralyzed, there may be a fracture of the petrous portion of the temporal bone, because these two nerves lies close together at this place. If the oculomotorius is paralyzed at the same time as the opposite side of the body, the lesion is in the peduncle. The trochlearis and trigeminus are affected least often of all. When they are paralyzed the basal site is likewise the first to be considered."

See also Böhne (44) and Dutoit (45).

As to paralysis accompanying meningitis, it is becoming increasingly evident that the paralysis of meningitis, and the same applies to tumor cases, is a matter of cerebrospinal pressure rather than infection *per se*. The beautifully scientific work of Kopetzky and Haynes (46) on meningitis may prove to be revolutionary so far as our conceptions on this subject are concerned. This applies as well to the accompanying choked disk.

Without going into detail of experience on this line, it is appropriate to present personal observations which seem to offer proof on this point. Within a brief space of time I had experience in two cases of meningitis of otitic origin.

The first of these was an abducens paralysis in which the paralysis of the sixth nerve was the initial sign of meningitis and persisted throughout. The paralysis occurred some time before streptococci were demonstrable in the spinal fluid.

The other case was one in which the slight papilla œdema and the paralysis of the abducens occurred simultaneously and

[1] Abducens paralysis is relatively common as a congenital anomaly, and it would seem more than logical to conclude that pressure or fracture occurs at this point not uncommonly in parturition.

examination of spinal fluid, which was found to be under pressure, gave the signs which Kopetzky[1] has proved to precede the more definite well-known symptoms of meningitis, namely, the absence of carbohydrate from the spinal fluid as evidenced by negative Benedikt copper reduction test, and a diminished alkalinity—an acidosis as shown by a modified Uffleman reagent. These tests for early diagnosis of meningitis were not made in the first case. In the second case I did a Haynes decompression operation—through the cisterna magna.

The interesting point to which I wish to call attention is the fact that in the second case, with the postoperative reduction of pressure in the cerebrospinal fluid, which was very high at the time of operation, there was a disappearance of the abducens paralysis, at the time of operation quite marked, and it did not recur during the eighteen days that the patient lived.

These two otitic cases present rather striking clinical evidence on this point, since the type of infection in the two was identical, and the site of infection the same; and while in the former case both the pressure symptoms and the abducens paralysis lasted throughout, in the latter the paralysis disappeared with the decompression and did not return.

HYSTERICAL PARALYSIS.

Hysterical paralysis has been ingenuously compared to sleep by Barnes (9). The central areas which subserve voluntary movements in consciousness are at rest during sleep. In hysteria these areas rest while the remainder of the brain is acting. On this basis, hysterical paralysis must have a cortical —third or high level—origin, and in the eyes will be evidenced by paralysis of conjugate movements, never by low level paralysis. Hysterical third, fourth, or sixth nerve paralysis is therefore impossible.

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[1] Kopetzky formulated the law that "the toxicity of the bacterial infection is dependent on the protein destruction which accompanies it, and to the disappearance of the available carbohydrate from the media in which the microbes are propagated," and second that "in the spinal fluid there is found evidence of acidosis in the varying degree of acidity, and the presence of different amounts of lactic acid."

NYSTAGMUS.

Other added proof on localization of control according to the foregoing table of binocular innervations is afforded by Maddox. The existence of areas of control for all subdivisions except 3 and 4 and 5 and 6 of the contrary movements and 11 and 12 of the parallel have had pathologic proof. It was left for Maddox to find clinical evidence of the existence of 3 and 4 and 11 and 12, and this he has done in a classical case of nystagmus of seesaw variety (47), which I take the liberty of introducing. The patient was a man of thirty-three who disclosed seesaw nystagmus when viewed from a distance, one eye always rising as the other fell, the movement being about 1*mm* in each eye. When the right eye was rising and the left eye falling, there was coincident conjugate parallel torsion to the left. When the left eye was rising and the right falling, there was conjugate parallel torsion to the right.

That the case strikingly confirms the existence of conjugate parallel torsional innervations 11 and 12 and the contrary vertical innervations 3 and 4 as tabulated, is asserted by Maddox, and this completes the evidence on this conception of binocular innervations, except subdivisions 5 and 6.

By our scheme of study of levels, these all doubtless lie in the forebrain—high level—for voluntary, and in the second level for reflex functioning.

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REPORT OF THE TRANSACTIONS OF THE SECTION ON OPHTHALMOLOGY OF THE NEW YORK ACADEMY OF MEDICINE.

By Dr. GEORGE H. BELL, Secretary.

MEETING OF OCTOBER 18, 1915. H. H. TYSON, IN THE CHAIR.

Dr. W. B. WEIDLER presented a case of **tubercle of choroid.**

A. R., æt. 18, clerk, with negative family history and good general health, stated that four weeks ago the right eye became red and the sight failed.

There was slight pericorneal injection, and posterior corneal deposits. To the nasal side of the nerve head there is a bluish-white area slightly pigmented with smaller highly refractive bodies about the margin. Wassermann negative; von Pirquet gave a slight positive reaction. After three months' treatment with tuberculin, vision improved from $\frac{20}{200}$ to $\frac{20}{40}$.

DISCUSSION: Dr. H. H. TYSON said that he had recently had an obstinate case of extensive tuberculous involvement of the choroid which did not show much improvement until after six months of tuberculin treatment, and at the end of nine months the case was slowly improving. He found that the general health improved faster than the ocular lesion under the tuberculin injections.

Dr. WEIDLER showed a case of **restoration of the socket.**

The patient, æt. 22, had had her left eye enucleated twelve years ago and had worn a glass eye until three years ago, when a small stump began to grow at the base of orbit which was probably a granuloma. This had been removed on three or four occasions but had always returned. The socket presents scars from the various operations and the lids are so retracted that the patient is unable to wear an eye.

The following operation was done May 3, 1915: After external canthotomy and removal of cicatricial tissue, freeing the lids, a large tongue-like flap, which was one-half an inch wide and about an inch long including the pedicle, was taken from the cheek and placed in the orbit and secured by several stitches. The orbit was then carefully packed with hot saline gauze so as to keep the flap from buckling on itself. The patient is now able to wear a full-sized eye, and there has not been any shrinking of the flap after six months.

DISCUSSION: Dr. SHINE thinks Dr. Weidler got a very good result, but thinks Wolff grafts would have been better on account of the scar.

Dr. WEIDLER also showed a case of **favus of scalp, eyelids, fingers, and toes.**

P. B., æt. 9, Italian, came to the Manhattan Eye and Ear Hospital, October 16, 1915, with almost complete loss of the hair of the scalp with deep scarring. There was complete absence of lashes on the upper and lower lids of both eyes, with very pronounced redness of the margins of the lids and free mucous discharge. There has been loss of the nails on both hands and feet. The condition has extended over a period of several years, and the patient has had no treatment for the disease. An examination of several hairs removed from the scalp shows the presence of spores.

Dr. F. W. SHINE showed a case of **cyst of ciliary body.**

The patient was a woman, aged 44; no history of eye trouble until two months ago, at which time patient accidentally discovered that she could not see out of the right eye. Vision = hand movements. Present condition: Lens cataractous and tilted on vertical axis so that the iris on temporal side is displaced forward. Nasally just behind the iris is what appears at first to be a pigmented growth, but on dilating pupil and on transillumination there is found to be a more or less translucent body of regular contour, and showing several parallel ridges or elevations. The root of the iris at this point is atrophic. X-ray negative. Tension normal.

Dr. F. W. IRWIN presented a case of **sarcoma of iris.** In a man, aged 40, a pigmented growth has been observed on the iris for six months without causing any symptoms. Viewed with a corneal microscope it stands out well and shows blood-

vessels. There have been several hemorrhages at this spot. Transillumination negative. Wassermann negative. The diagnosis was made of sarcoma of iris, which very probably encroaches on the ciliary body and an enucleation will be done.

DISCUSSION: Dr. CURTIN thought it might be well to do an iridectomy and have the mass examined; and if it proved to be sarcoma to enucleate the eye at once.

Dr. WEIDLER advised enucleation at once.

Dr. H. H. TYSON stated that about ten years ago he presented before this Section a case of sarcoma of the iris resembling Dr. Irwin's case, excepting that the growth was much smaller in appearance on the anterior surface of the iris. Vision $\frac{20}{20}$. Patient complained only of an occasional blurring of vision, and desired glasses. The blurring was due to small hemorrhages. After seeking advice from many ophthalmologists, he finally consented to the enucleation of the affected eye. Microscopic section showed that the growth had invaded the posterior surface of the iris extensively and involved the ligamentum pectinatum. The patient is still alive and enjoying good health. He advises early enucleation in Dr. Irwin's case, and is opposed to iridectomy, as the latter would be more liable to induce metastasis.

Dr. LINN EMERSON read a paper on **The relative importance of the fitting of glasses in ophthalmic practice.**

Dr. Emerson said few doctors and fewer patients realize that the fitting of eyeglasses is the 33d degree of ophthalmic practice. In 1874, Dr. William Thompson of Philadelphia discovered that the accurate correction of ametropia cured headache, insomnia, vertigo, nausea, failure of general health, and various nervous and psychic ailments. These facts he communicated to Dr. S. Weir Mitchell, to whom the credit must be given for having been the first to write at any length on the subject of "Headache and Eyestrain." It is now generally conceded by most oculists that 90 per cent. of sick headaches are due to eyestrain, and because a patient has had glasses and has not been relieved is no sign that eyestrain is not the cause of the trouble. The oculist who fails to cure these cases and considers his more successful colleague an extremist or hobby rider, would do well to practice introspection.

Dr. Emerson in concluding said:

1st. While formerly the fitting of glasses was relegated to the maker of spectacles as beneath the dignity of the ophthalmologist, at present it constitutes the larger part of ophthalmic practice.

2d. While formerly it was thought unnecessary or even undesirable to use a cycloplegic, it is now conceded by most ophthalmologists that in patients under thirty-five years of age the use of a cycloplegic is a necessity to accurate fitting.

3d. The ophthalmologist who persists in doing refraction without a cycloplegic is, in the eyes of the patient, and often justly so, no better than the optician.

4th. The day of the superficial, careless, hurried, slipshod fitting is past, and to be successful and relieve the patient careful and accurate refraction is necessary.

5th. The oculist who tells a patient he has not astigmatism enough to justify its correction is behind the times, and should follow Elbert Hubbard's advice to "get in line."

6th. At least 50 per cent. of headaches of civilized humanity are due to eyestrain, and the general practitioner who treats a case of recurrent headache without having the patient's refractive condition investigated, is as remiss as though he neglected blood pressure or urinalysis.

7th. As oculists we should avail ourselves of every opportunity to impress on the general practitioner and the public the fact that eyestrain is a frequent cause of headache, insomnia, vertigo, nausea, failure of general health, and various nervous and psychic ailments.

8th. The unskillful and careless fitting by oculists in the past is in a large measure responsible for the presence among us of eyesight specialist or optometrist; and in many instances his fitting is but little worse than the oculist who does not find it necessary to employ a cycloplegic.

9th. Badly adjusted glasses are as often a cause of failure to relieve as improper fitting. Of the glasses worn to-day, less than 25 per cent. are accurately fitted and properly adjusted.

10th. The accurate fitting of glasses to the young will reduce the ophthalmic surgery necessary in the later decades of life by at least 10 per cent.

11th. The way to eliminate the optometrist is to rise to the

occasion, and do better work than he does. Oculists whose fitting is of the optometrist's standard should not complain of his presence. The legislators will not listen to us until we show by our improved work that we exceed them in skill.

Dr. H. H. TYSON, in discussing the action of the combined solution of homatropia and cocaine in the eye, said that the advantage of the combination existed in the fact that a greater dilatation of the pupil was obtained, which enabled an easier retinoscopic examination, and incidentally permitted a more thorough examination of the anterior parts of the fundus.

MEETING OF NOVEMBER 15, 1915.

Dr. LINN EMERSON showed a case of **foreign body imbedded in the optic nerve: not removed; globe preserved.**

The patient, a man fifty years of age, while at work got a piece of steel in his eye. When first seen, two days after the accident, he had iritis and œdema of conjunctiva. He was taken to the Manhattan Eye, Ear, Nose, and Throat Hospital for an X-ray, which showed the piece of steel imbedded in the optic nerve. The magnet had no effect on the foreign body. The eye is now quiet. The tension is minus. V. = 0. Dr. Emerson wanted to know the opinion of the Section as to whether or not the eye should be enucleated. He is of the opinion that it should be left alone.

Dr. L. W. CRIGLER thought it would be well to take another X-ray picture to see if the foreign body is still imbedded in the nerve.

Dr. H. H. TYSON stated that, considering the eye was blind with diminished tension, and the known fact that it contained a foreign body as shown by the X-ray plate, he believed enucleation to be the proper procedure in order properly to conserve the vision and safety of the other eye; and that if enucleation was not permitted now, careful and frequent examination should be made, and upon the first appearance of any suspicious symptoms of irritation in the other eye the enucleation of the injured eye should be insisted upon, in justice to the patient and the surgeon.

Dr. H. H. TYSON presented a case of **disciform keratitis.**

The patient was a young man, seventeen years of age, with keratitis disciformis in his left eye. The disk-shaped opacity

in the center of his cornea had the characteristic uneven and striped surface, with no history or signs of inflammation. Vision, O. D., $\frac{20}{40}$; O. S., $\frac{20}{70}$. Wassermann, negative; von Pirquet, markedly positive. Local reaction in eye after injection of *1mg.* T. O. Temperature, 99.8° F. Fuchs states that these cases are caused by ectogenic infection, followed by inflammatory infiltration, and scar tissue formation as an end result. This case was the fourth examined by Dr. Tyson during the past three years, all showing markedly positive tuberculous reactions; one case improving under tuberculous therapy. He is inclined to the opinion that tuberculosis is either an exciting or a contributory cause in this condition.

Dr. GEORGE H. BELL reported a case of **intraocular foreign body (steel).** The man, aged twenty-one, had been treated for four weeks before consulting Dr. Bell. When first seen, he had a scar on the cornea and a traumatic cataract. The X-ray showed a foreign body in the vitreous about *4mm* behind the lens. The foreign body and the cataract were both extracted without doing an iridectomy. The patient now has a round and central pupil, and with correction has a vision of $\frac{20}{30}$.

Dr. ANNA W. WILLIAMS read a paper on **purulent conjunctivitis in infants under two months of age,** which appears in this issue of these ARCHIVES.

In discussing the paper, Dr. H. H. TYSON inquired as to whether the source of the infection was discovered in those cases where the xerosis bacillus predominated, and as to the prophylactic used in most of the lying-in hospitals. He had had the opportunity of examining a number of cases of purulent conjunctivitis in the newborn in a maternity hospital, in which the xerosis bacillus was considered to be the cause of the condition and the origin of the infection was not definitely determined. This institution was using argyrol solution as a prophylactic. He thought it advisable to follow the Credé method. The fact that a majority of the cases of purulent conjunctivitis in the newborn were caused by other pathogenic organisms than the gonococci should not lead us to be less careful in our prophylaxis, but should stimulate us to greater care on account of the danger of infection being more widespread, and a possibility of more frequent occurrence with just as disastrous results.

REPORT OF THE TRANSACTIONS OF THE SECTION ON OPHTHALMOLOGY OF THE ROYAL SOCIETY OF MEDICINE.

By C. DEVEREUX MARSHALL, F.R.C.S., London.

The ordinary meeting of the Section was held on Wednesday, November 3d, Mr. Priestley Smith, President of the Section, in the Chair.

The President made sympathetic reference to the recent death, after an operation, of Mr. George Coats, of whose scientific contributions he spoke very highly. Mr. Coats acted as Honorary Secretary to the last Congress of the Ophthalmological Society, and carried out the duties with great thoroughness and every courtesy.

Captain Ormond showed a case which exhibited associated movements of jaw and eyelid, and discussed the probable ætiology, based on Bishop Harman's work.

Dr. C. W. Daniels contributed a paper entitled **Eye lesions as a point of importance in directing suspicion to possible trypanosome infection.** He pointed out the practical importance to be attached to the eye lesions as a diagnostic sign of infection with trypanosomes, inasmuch as, in some cases, they first determined the patient seeking medical advice. The common manifestation was an iridocyclitis, while a pronounced œdema of the lower eyelids was not rare. Choroiditis was less frequent. The incidence of the eye lesions stood in direct relationship with the severity of the infection. Thus, in Rhodesian trypanosomiasis the eyes were affected in 83.3%; in Nigeria cases in 40%, and in cases from Uganda and other parts of tropical Africa in 18.7%. The mortality from the disease in those three regions was, respectively, 100%, 30%, and 18%.

Mr. LESLIE PATON, who read the paper in the author's absence, discussed the matter, and referred to Mr. Browning's work in regard to the blood changes found in sympathetic ophthalmia.

Mr. SYDNEY STEPHENSON read a communication entitled **A fallacy in the diagnosis of glioma retinæ,** describing the clinical and pathological details of two cases, both in young children, whose blind eye was excised under the impression that it contained glioma. It was found, however, that it was affected with the rare disease retinitis exudativa (retinitis hæmorrhagica externa). The author laid stress on this possible source of confusion.

The paper was discussed by the President, Mr. TREACHER COLLINS (who suggested useful diagnostic signs for differentiation), Mr. STEPHEN MAYOU, Mr. J. B. LAWFORD, Mr. JESSOP, Mr. ZORAB, and Captain ORMOND.

Dr. A. HUGH THOMPSON read a paper on **Late results of the operative treatment of high myopia.** He said that five years ago he published some notes based on his experience of twenty myopic eyes, the clear lenses of which he had needled with the object of making the refraction approximately emmetropic. His purpose was to relate now his further experience of such of those cases as he had been able to keep in touch with. During the last five years, in common with many others, he had been chary about recommending this operation, though he still maintained that in carefully selected cases the treatment was a good one. As one could not be certain about the future of these eyes, the procedure should not be pressed upon any patient, for one could not promise cure, and the eye was left just as liable as ever to those destructive processes which attacked highly myopic eyes. The degree most suitable for operation was 16 to 22 diopters. In young children, whose myopia was certainly progressive, a limit of 14 D. might well be assigned. With a myopia of more than 22 D., the chance of the eye remaining free from destructive processes was small. His method had been a free needling of the lens capsule, after which it was necessary to watch the patient carefully day by day, and in the event of a rise of tension, let out the lens matter by means of a keratome incision through the cornea. Should a secondary membrane form some

years after, this must be needled. He had been able to follow up fourteen cases, during periods of five to fifteen years. Only one was operated upon in both eyes, and he did not think the advantage justified the double operation. In all the fifteen eyes, the result of the operation was good, and the subsequent results he tabulated in detail. Two-thirds of the cases, after an average period of eight years, had materially better vision than before the operation. In not one of them had he to deal with a detachment of the retina as a sequela to the operation, though it was well known to occur occasionally. He did not think the operative treatment of high myopia tended to counteract the increase in the long axis of the eyeball, though obviously the removal of the lens had the effect of diminishing the result of the lengthening of the axis of the eyeball on the refraction, by about one-half.

An interesting discussion ensued, which was taken part in by Mr. HARRISON BUTLER, Mr. R. COULTER, Mr. ARNOLD LAWSON, Mr. W. H. JESSOP, Mr. HOLMES SPICER, Mr. ZORAB, and the President.